IDSP

Industrial
Disease
Standards
Panel

92-07878/

IDSP

Fire Fighters in Metropolitan Toronto:
Summary of the Mortality Study

Kristan A. L'Abbé and George A. Tomlinson
April 24, 1992

Industrial Disease Standards Panel
IDSP Occasional Paper
Toronto, Ontario

Industrial Disease Standards Panel

In 1985 the Ontario legislature established the Industrial Disease Standards Panel (IDSP) to investigate and identify diseases related to work. The Panel is independent of both the Ministry of Labour and the Workers' Compensation Board. At the end of each fiscal year the WCB reimburses the Ministry for the Panel's expenditures.

The Panel's authority flows from section 95 of the *Workers' Compensation Act* and its functions are set out as follows:

(8) (a) to investigate possible industrial diseases;

(b) to make findings as to whether a probable connection exists between a disease and an industrial process, trade or occupation in Ontario;

(c) to create, develop and revise criteria for the evaluation of claims respecting industrial diseases; and

(d) to advise on eligibility rules regarding compensation for claims.

Decisions of the Panel are made by its members who represent labour, management, scientific, medical and community interests. Once the Panel makes a finding, the WCB is required to publish the Panel's report in the Ontario Gazette and solicit comments from interested parties. After considering the submissions the WCB Board of Directors decide if the Panel's recommendations are to be implemented, amended or rejected.

To assist with its work the Panel has a small staff of researchers, analysts and support people. In addition to its own staff, the Panel relies heavily on the advice of outside experts in science, medicine and law, as well as input from the parties of interest.

Canadian Cataloguing in Publication Data

L'Abbé, Kristan A.
Fire fighters in Metropolitan Toronto

(IDSP occasional paper)
Includes bibliographical references.
ISBN 0-7729-9794-2

1. Fire fighters—Ontario—Toronto Metropolitan Area—Mortality. I. Tomlinson, George, 1964- . II. Ontario. Industrial Disease Standards Panel. III. Title. IV. Series.

HB1323.P82C35 1992 304.6'4'0883633 C92-092552-9

Additional copies of this publication are available in writing:
Industrial Disease Standards Panel
69 Yonge Street, Suite 1004
Toronto, Ontario M5E 1K3
(416) 327-4156

Acknowledgements

We wish to thank all fire department employees who were enroled in this study, and we are grateful for the cooperation of unions and management in this effort. In particular, we thank Chief Joe Gibson, Dr. J.T. Bates, Evelyn Funston, and Jim Pullen. The protocol was initiated by Dr. Lesbia Smith. The data collection phase was greatly assisted by four enthusiastic research assistants, Wendy Findlay, Jani Gendron, Jodi Gendron, and Debbie Morin. We also acknowledge the contributions of Statistics Canada (Martha Fair, Maureen Carpenter, and Pierre Lalonde), the provincial and territorial Registrars of Vital Statistics, the Ontario Cancer Treatment and Research Foundation (Eric Holowaty and Darlene Dale), peer reviewers, and the staff of the Industrial Disease Standards Panel.

Introduction

The Industrial Disease Standards Panel (IDSP) undertook a study of mortality among fire fighters after the unions representing fire fighters made submissions to the Panel concerning excess cardiovascular disease among their members.

Purpose of the Study

There is conflicting evidence of increased risk of cardiovascular disease among fire fighters. Two previous studies among fire fighters in the City of Toronto have shown slight excesses in cardiovascular mortality (Mastromatteo, 1959; Bates, 1987), and one recent study of fire fighters in Hawaii showed a borderline elevation (Grimes et al., 1991). However, numerous other studies carried out in Australia, Canada and the United States have not found an overall increase in cardiovascular mortality (Musk et al., 1978; Eliopulos et al., 1984; Vena and Fiedler, 1987; Beaumont et al., 1991; Demers et al., 1992; Guidotti, 1992). But within some of these studies, sub-groups with either long duration of employment or time since first exposure have exhibited excess risk (Vena and Fiedler, 1987; Demers et al., 1992).

Several studies of fire fighters have demonstrated increased risk for cancer at various sites. A report that combines the results from several studies suggests that fire fighters experience excess mortality from brain cancer and possibly multiple myeloma (Howe and Burch, 1990).

Because of this conflicting evidence, this study was designed to investigate whether there is excess mortality among Metropolitan Toronto fire fighters, with particular attention to cardiovascular disease. Excess mortality is said to occur when there are more deaths in the study population than would be expected based on death rates in a reference population.

This report is intended as a descriptive overview of the main results of the study. The full scientific report ("Scientific Report of the Mortality Study of Fire Fighters in Metropolitan Toronto") is available upon request.

Definition of the Cohort

A retrospective cohort study was conducted, with subjects recruited from all six fire departments within Metropolitan Toronto: the cities of Toronto, North York, Scarborough, Etobicoke and York and the Borough of East York. Data was collected on 5,995 employees of fire departments.

Subsequently excluded were (Table 1):

- females
- those who had worked less than six months
- those not known to be alive on or after Jan. 1, 1950
- those without a known birth date
- those with no known hire date
- those not currently employed but had no known termination date (excluded from duration of employment analysis only).

TABLE 1
Exclusions and Number of Firefighters Removed Due to Each Exclusion

Size of cohort before exclusion	*Reason for exclusion*	*Number excluded*
5995	Duplicate entries	28
5967	Females	146
5821	Known dead before 1 Jan 1950	65
5756	Not known alive after 1 Jan 1950	58
5698	Unknown birth dates	48
5650	Unknown hire	24
5626	Less than 6 months' employment	211
5415	Aged 85 before follow-up began	1
5414	Unknown termination dates	41
Final size of cohort for principle analysis		5414
Size of cohort for analyses using duration of employment		5373

Following these exclusions, 5414 individuals were included in the analysis. Their mortality experience was analyzed over the period from January 1, 1950 to December 31, 1989.

Methods

Records of employment histories were located in fire department headquarters, personnel offices and archives. All departments maintain paper or card files to some extent, some use microfilm or microfiche and recently some have been storing information on computer. A standard data collection form was developed to facilitate direct entry of information into a computer database. Four data abstractors were briefed on issues of confidentiality and were required to take an oath of secrecy.

The resulting data file containing information on each fire department employee for whom records were available was sent to Statistics Canada (SC). Through record linkage with the National Mortality Data Base (NMDB), SC determined the number of deaths in the cohort, and where applicable, the cause and date of death was added to the file. This system is a very reliable means of determining occurrence and cause of death for residents of Canada when it is provided with the full name and

date of birth of an individual (Shannon et al., 1989). SC then removed all personal identifiers from this file and returned it to the IDSP staff for statistical analysis.

Mortality of the cohort specific to age, calendar period, and cause was compared to that of the Ontario male population over the period 1950-1989. Subjects began to contribute person-years at risk on January 1, 1950 or their first day of employment, whichever was later, and ceased to contribute person-years on their date of death, upon reaching age 85 or on December 31, 1989, whichever was earlier. The protocol called for censoring at age 85 on the grounds that certification of cause of death in elderly people is less reliable.

The number of deaths expected from a particular cause was computed by multiplying the person-years in each age and calendar period stratum by the cause-specific Ontario mortality rates for that stratum. The number of deaths observed in the cohort is compared to the number of expected deaths by taking the ratio of observed to expected, and when multiplied by 100 this yields the standardized mortality ratio (SMR):

$$\frac{\text{observed deaths}}{\text{expected deaths}} \times 100 = \text{SMR}$$

An SMR greater than 100 may indicate an excess risk of death due to a particular cause. The statistical significance of an apparent excess is judged using a 95% confidence interval (CI) (Breslow and Day, 1987). When the SMR is greater than 100 and the CI excludes 100, there is said to be a statistically significant excess in mortality in the cohort. Likewise, when the SMR is less than 100 and the CI excludes 100, there is said to be a statistically significant deficit in mortality in the cohort. In these situations, chance as an explanation of the results is excluded 19 out of 20 times. Elevated, although not statistically significant (i.e., the 95% CI includes 100), mortality risks are judged in this report as SMRs over 130 with 4 or more deaths.

Information on deaths which were certified to any type of cancer was sent to the Ontario Cancer Treatment and Research Foundation (OCTRF). From records which date back as far as 1964, they returned confirmation of the cause of death and information on cancer incidence, including histological type and date of diagnosis when available.

Results

Descriptive statistics for the 5414 fire department employees are seen in Table 2. At the end of follow-up, 55% of the subjects were classified as current employees, while the remaining 45% were retired or terminated.

TABLE 2
Descriptive Statistics

Variable	*N*	*Minimum*	*Maximum*	*Mean*	*Std Dev*
Follow-up	5414	0.38	40.00	21.46	11.79
YSFE	5414	0.57	67.71	24.38	14.59
Agehire	5414	16.40	61.52	25.28	3.79
Agedeath	837	22.77	95.83	63.29	14.96
DUR	5373	0.50	54.75	19.89	11.99

Over the period of the study, there were 837 deaths in the cohort and 60 deaths were excluded from the analysis because they occurred after age 85. This left 777 deaths, of which 29% occurred in current workers or within one month of termination of employment, 61% more than two years after termination, and the remaining 10% between these two intervals. The mean age at death was 63 years.

The results below begin by discussing all-cause mortality, and continue with circulatory system, cancer, and respiratory system and digestive system diseases. Summaries of the analyses are seen in the Tables attached, but these are generally not referred to in the text. As well as presenting a summary of the results of this study, we have attempted to evaluate our findings in relation to the body of knowledge available. Salient points are discussed below according to cause.

Overall Mortality

Cause-specific mortality for the total cohort is presented in Table 3. The all-cause SMR is 94 (95% CI: 88-101). As expected, mortality risk among current workers is very low (SMR=56), and that among terminated workers is higher (SMR=119).

Most of the major groups of causes of death, including circulatory system diseases and cancer, have SMRs of about 100, indicating no excess overall in comparison with the general population.

Most of the major groups of causes of death, including circulatory system diseases and cancer, have SMRs of about 100, indicating no excess overall in comparison with the general population. The two exceptions among the major groups of causes of death are infective and parasitic diseases, and endocrine, nutritional and metabolic diseases which exhibit statistically significant deficits in mortality. Among specific causes of death, two causes exhibit statistically significant deficits in mortality: diabetes mellitus and chronic rheumatic heart disease, as these are conditions which may cause an individual not to seek employment as a fire fighter. These will not be discussed further.

TABLE 3

Mortality by Detailed Cause for Total Cohort

Cause of death (ICD9)	*OBS*	*EXP*	*SMR*	*95% CI*
All Causes (000-999)	777	823.50	94	(88-101)
Infective, Parasitic Diseases (1-39)	1	7.21	14	(0-77)
All Malignant Neoplasms (140-208)	199	190.39	105	(90-120)
Pharynx (146-149)	4	2.87	139	(38-357)
Oesophagus (150)	2	5.04	40	(5-143)
Stomach (151)	7	13.79	51	(20-105)
Colon (153)	11	18.25	60	(30-108)
Rectum and rectosigmoid junction (154)	13	7.60	171	(91-293)
Liver and bile ducts (155)	2	2.37	84	(10-305)
Pancreas (157)	14	9.98	140	(77-235)
Larynx (161)	1	2.70	37	(1-206)
Trachea, bronchus & lung (162)	54	57.08	95	(71-123)
Malignant melanoma (172)	2	2.76	72	(9-262)
Prostate (185)	16	12.09	132	(76-215)
Testis (186)	3	1.22	246	(51-719)
Bladder (188)	7	5.49	128	(51-263)
Kidney and ureter (189)	2	4.63	43	(5-156)
Brain and other nervous system (191-192)	14	7.00	200	(109-336)
Lymphatic & hematopoietic tissue (200-208)	18	18.39	98	(58-155)
Lymphosarcoma (200)	3	1.48	203	(42-592)
Hodgkin's disease (201)	1	2.19	46	(1-254)
Multiple myeloma (203)	1	2.59	39	(1-215)
Lymphatic leukaemia (204)	4	2.11	190	(52-485)
Myeloid leukaemia (205)	4	3.35	119	(33-306)
Other	5	6.67	75	(24-175)
Other malignant (195-199)	20	7.08	282	(172-436)
Endocrine, Nutritional Diseases (240-279)	5	14.61	34	(11-80)
Circulatory System (390-459)	384	387.79	99	(89-109)
Ischemic heart disease (410-414, 429.2)	289	278.38	104	(92-117)
Acute myocardial infarction (410)	205	191.65	107	(93-122)
Cerebrovascular disease (430-438)	41	54.02	76	(55-103)
Arteriosclerosis (440-448)	24	16.98	141	(91-210)
Aortic aneurysm (441)	19	8.40	226	(136-353)
Diseases of veins, etc. (415, 451-459)	8	4.78	167	(72-330)
Respiratory System (460-519)	45	46.12	98	(71-131)
Chronic bronchitis, emphysema, asthma (490-493, 496)	28	21.47	130	(87-189)
Digestive System (520-579)	45	36.85	122	(89-163)
Gallbladder diseases ( )	5	1.69	296	(96-690)
Genitourinary System (580-629)	11	10.41	106	(53-189)
Symptoms/Ill-Defined (780-799)	1	6.06	17	(0-92)
External Causes (E800-999)	70	100.72	69	(54-88)
Motor vehicle traffic accidents (810-825)	22	31.10	71	(44-107)
Accidental falls (880-888)	9	7.82	115	(53-219)
Accidents caused by fire & flames (890-899)	4	3.23	124	(34-317)
Suicide (950-959)	18	26.68	67	(40-107)
????Other Known Causes	16	22.81	70	( )

Statistically significant excesses are seen for three specific causes of death:
(1) cancer of the brain and other nervous system tumours,
(2) "other" malignant neoplasms, and,
(3) aortic aneurysm.
These will be discussed extensively below.

The Healthy Worker Effect

When this study was designed, there was a concern that the bias known as the healthy worker effect would mask some important risks. This bias can occur when the mortality experience of a working population is compared to that of the general population, which comprises both those who work and those who do not or cannot work. The working population, being healthier, is expected to have a lower mortality rate for many causes, so there is sometimes a problem in determining what exactly constitutes an excess risk for the working population. Fire fighting is unlike most occupations in that to become a fire fighter, one must pass a medical examination, an aptitude test and tests of physical skill. The six fire departments in this study all had approximately the same entrance requirements. Although the requirements were not so stringent in the past, to be hired, one has always had to be physically fit. It does not seem unreasonable to assume that at least at the start of his career, the fire fighter is much healthier than the average person in the general population.

In this study, a strong healthy worker effect is evident for those under 40 years of age. In other words, fire fighters under 40 die less frequently than comparable members of the general population. However, by the time a fire fighter reaches 40, he is no longer at a reduced risk of death when compared to the general population. Furthermore, all-cause mortality shows an increasing trend in SMRs as years since first exposure (YSFE) increase. Analysis by 10 year periods indicates that a strong healthy worker effect is evident for those with less than 10 YSFE, but not evident for those workers with more than 10 YSFE. In other words, although the fire fighters experience a lower mortality rate than comparable members of the general population, this is only so for approximately the first ten years since start of employment. After this ten year period, for many causes of death their mortality experience is similar to or higher than that of the general population.

Although the fire fighters experience a lower mortality rate than comparable members of the general population, this is only so for approximately the first ten years since start of employment. After this ten year period, for many causes of death their mortality experience is similar to or higher than that of the general population.

In anticipation of an overall healthy worker effect in these results, the protocol for this study provided for a comparison of the fire fighters to another working population, the Northern Ontario Miners' Cohort. The comparison to miners would have been subject to some severe limitations for several reasons. In any event, because no strong healthy worker effect overall is evident in this study, this comparison is not necessary. Because of

the pattern of increasing SMRs with increasing YSFE, there is some difficulty in interpreting the SMRs. For some causes an SMR of 100 may be an excess relative to an SMR of about 85 for all-cause mortality that has been shown for several working populations (Meijers et al., 1989). Therefore, depending on what one judges as the most appropriate comparison group for the fire fighters, the real magnitude of some of the risks seen here may be greater than the SMRs indicate.

In the other cohort study of Canadian fire fighters, with the Albertan population as a reference group, a healthy worker effect was also not seen (Guidotti, 1992). It has been reported, however, in several studies of U.S. fire fighters (Beaumont et al., 1991; Demers et al., 1992, for example). Dr. Guidotti explains that a healthy worker effect is not seen in his study because few subjects are lost to follow-up (as in this study), there is a long follow-up period (as in this study), and the use of a provincial instead of national comparison may reduce the effect since Alberta males are generally healthier than the national average.

The Survivor Effect

Another effect that could mask the results is the survivor effect. Because adequate health is required for continuing employment and unwell workers may selectively leave the fire department before 30 years of employment, it may be that only the healthiest workers continue to be employed long-term. If duration is the only proxy for exposure and there is a survivor effect, a true exposure-response relationship over the range of employment duration may be masked (Robins, 1987). This may explain in part, for example, the increases in SMRs seen until 30+ years' duration, and the decline seen after this point for some causes of death. The healthy worker effect is generally more pronounced for cardiovascular disease than for cancer causes of death.

Circulatory System

This overall group of causes of death ("Major cardiovascular diseases") is comprised of four sub-groups, with the first accounting for the majority of deaths: ischaemic heart disease, cerebrovascular disease, arteriosclerosis, and diseases of the veins, lymphatics and other circulatory system. Within ischaemic heart disease, the largest sub-group is acute myocardial infarction, and within arteriosclerosis, the largest sub-group is aortic aneurysm.

Major cardiovascular diseases (SMR=99)

The group of causes labelled "major cardiovascular diseases" was responsible for almost half of the deaths in this study, as it is for the general population. The SMR hovers around 100 across all calendar periods and for all categories of YSFE and age at risk. There is no obvious pattern according to duration of employ-

ment. While risk is low among current workers (SMR=62), it is higher among terminated workers (SMR=117). Within this overall cause of death category, ischaemic heart disease accounted for a majority of the deaths, again with no apparent excess, and also displayed no pattern of risk with the temporal or exposure variables.

This differs from the findings of Mastromatteo (1959) who reported an excess in City of Toronto fire fighters for deaths occurring between 1921 and 1953 from cardiovascular-renal diseases. This overlaps with the present study by four years of follow-up, a period in which there is no apparent excess in our study.

Coronary heart disease (CHD) was also elevated in the study by Bates (1987) of fire fighters in the City of Toronto who worked six years or more. He compared mortality rates to those for males in the City only. Follow-up was from 1949 through 1984, and of 52 deaths known through pension records, 47 death certificates were obtained and examined for any circulatory system cause of death. For the remaining deaths, cause was abstracted from newspaper or coroner reports. The resulting SMR for CHD was 173 based on 21 deaths. A possible explanation for these discrepant results is the different methodologies (different inclusion criteria, method of death enumeration, method of cause of death coding, and comparison populations) used by Bates and in the present study. It is important to note that in an unpublished survey of smoking among fire fighters in 1980 in the City of Toronto, about one-third smoked. This is approximately the same as the proportion of males in the general population who smoked, and smoking can therefore be discounted as an explanation of the increased risk seen for CHD. Dr. Bates is continuing work on this issue and is looking at the possible contribution of cholesterol levels and hypertension. Two studies have shown increasing SMRs for circulatory system diseases with increasing years of work (Vena and Fiedler, 1987; Heyer et al., 1990, and updated in Demers et al., 1992). Guidotti found no association with duration of employment for this cause (Guidotti, 1992).

A recent review discusses the possible role of occupational factors in the development of cardiovascular diseases (Kristensen 1989a and b). Some non-chemical occupational factors which could contribute to cardiovascular disease risk in fire fighters are sudden exertion during waiting or sleeping periods, shift work, environmental stress (heat, cold, noise), physical stress (from heavy equipment, prolonged work), and emotional stress (critical incidents such as witnessing fire victims, response to emergencies, responsibility for the lives of others). Substances which may potentiate a cardiovascular reaction in fire fighters include carbon monoxide and dioxide, acrolein, benzene, cyanide, hydrogen chloride, parti-culates, sulphur oxides, and organic solvents.

Although the overall SMRs for acute myocardial infarction are not statistically remarkable, they are higher than would be expected from a population whose work requires a degree of physical fitness higher than that of the general population.

Myocardial infarction (SMR=107)
The SMRs do not vary substantially from 100 over the range of YSFE, while analysis by age indicates that the risk is slightly higher for those under 60 years. There is no apparent trend with duration of employment. Again, risk is low among current workers (SMR=71) and high among terminated workers (SMR=129). Although the overall SMRs for acute myocardial infarction are not statistically remarkable, they are higher than would be expected from a population whose work requires a degree of physical fitness higher than that of the general population.

For ischaemic heart disease as a whole, Hansen (1990) reports a slight excess, while most cohort studies of fire fighters, which use general population comparisons, do not show any evidence of an increased mortality risk for ischaemic heart disease or myocardial infarction.

Arteriosclerosis (SMR=141)
The excess seen for arteriosclerosis does not exhibit any pattern according to YSFE. Risk is slightly elevated in younger men and even higher in older men. Risk is relatively high and constant across all duration categories.

Aortic aneurysm (SMR=226)
The majority of deaths attributed to the larger category of arteriosclerosis are due to aortic aneurysm. Of the 19 deaths, no death occurred before 20 YSFE, but at 20-30 YSFE the SMR rises to 303 and at 40-49 YSFE, there is a statistically significant SMR. Risk is higher among those over age 60 than in those under age 60. Risk also increases from the first to second category of duration of employment, then remains stable. Although one death occurred in a fire fighter with 1-4 years of employment, all others occurred in those with 15 or more years of employment and risk is elevated only for terminated workers.

Smoking, hypertension, diabetes, atherosclerosis, trauma and stress have been identified as possible risk factors (Lindsay and Hurst, 1979).

Diseases of the veins, lymphatics and other circulatory system (SMR=167)
No death occurred before 10 YSFE or after 50 YSFE, or in those with less than 15 years employment. Risk is elevated only for those with 15-29 years duration of employment, but is identical for current and terminated workers.

Cancer

The results for this section are organized by cancer site. For five cancer sites, a review has combined results from six cohort stud-

ies of fire fighters (Howe and Burch, 1990). Since that publication, four additional cohort studies of fire fighters are available which present results on the sites of cancer covered in the review (Tornling et al., 1990; Beaumont et al., 1991; Demers et al., 1992; Guidotti, 1992). For three of these cancer types (colon, malignant melanoma, and multiple myeloma), results are unremarkable in the present study, and are not covered in this summary report. For the other two sites (brain and other central nervous system tumours, and lung cancer), we attempt here to combine our study results with all results available to us.

With 13 deaths from this site, there is statistically significant excess mortality.

Cancer of the brain and other nervous system (SMR=200) With 13 deaths from this site, there is statistically significant excess mortality (SMR=200, 95% CI: 109-336). Excess risk is seen in all calendar periods examined except 1975 through 1984. Large excesses are also seen for 0-19 YSFE and 40-59 YSFE, but not for 20-39 YSFE. The level of risk is identical for relatively young and older fire fighters. Breakdown by five year groupings of duration of employment revealed that no deaths occurred in those with less than 5 years of employment, but that the risk is statistically significant at 5 to 9 years duration. According to broad categories of employment duration, risk exhibits a bimodal pattern, showing an excess for employees working less than 15 years and more than thirty years, but not differing from that in the general population in the intervening period.

According to information received from the OCTRF, one subject whose primary cause of death was certified as a different cancer site death was recorded as an incident case of brain cancer in the Ontario Cancer Registry. The certification of the death can be thought of as an error, in that the other site was most likely secondary to the primary brain cancer (Dr. Holowaty, OCTRF, personal communication). The histological type was glioma. The remaining nine subjects who died from brain cancer after 1963 were registered as incident brain cancer cases at the OCTRF and six of these were histologically confirmed, all as gliomas.

[Within the total cohort enumerated, there was one additional death certified as cancer of the brain and other nervous system, but the subject was excluded from the analysis based on the criterion that the death occurred before 1950.]

When the results of the present study are combined with the previous studies' results, the overall SMR is 164 (95% CI: 126-210). In addition, two recent studies, not included in the combined results because they are not cohort studies, have reported a possible increased risk of brain cancer in fire fighters.

When the results of the present study are combined with the previous studies' results, the overall SMR is 164 (95% CI: 126-210). In addition, two recent studies, not included in the combined results because they are not cohort studies, have reported a possible increased risk of brain cancer in fire fighters. A study of fire fighters in Hawaii produced a proportionate mortality ratio of 378 based on 3 deaths (Grimes, 1990), and a case-control study in Massachusetts gave a standardized morbidity odds ratio

of 86 when the state pop-ulation was used as a reference, and 152 when police were used as a reference (Sama et al., 1990).

A comprehensive review of brain cancer and occupational risk factors found numerous studies showing increased risk of brain cancer in a wide variety of occupational groups (Thomas and Wax- weiler, 1986). The lack of exposure information in most of these studies makes it difficult to determine exactly what any of them might have in common with fire fighting. However, the authors mention, among other substances, vinyl chloride, formaldehyde, acrylonitrile, polycyclic aromatic hydrocarbons, diesel and other motor fuel exhausts, trichloroethylene, asbestos and solvents as exposures that are prevalent among many of the occupational groups with increased risk of brain cancer. A recent study has demonstrated a brain cancer excess in workers exposed to poly-chlorinated biphenyl (Sinks et al., 1991).

Other malignant neoplasms (SMR=282)
With 20 deaths from this cause, statistically significant excess mortality is evident. There is some excess risk apparent in all calendar periods. No death occurred before 10 YSFE, and after this time the SMR increases as YSFE increases, until 50+ YSFE.

According to age at risk, there are two periods of statistically significant excess: at ages 50-59 and 70-85. According to five year periods of employment, there are two statistically significant excesses, at 10-14 and 35+ years. Although one death occurs in a worker with 1-4 years of employment, all others occur in those with 10 or more years of employment. This cause of death is elevated among current and terminated workers alike.

Of the 20 "other malignant neoplasm" deaths, 17 were diagnosed with cancer after 1963. The OCTRF certified 11 of these subjects as "ill-defined site" incident cases. Of the remaining six subjects, two had cancer incidence of the same site and the other four had cancer at different sites.

Cancer of the pharynx (SMR=139)
There is a suggestive increase in mortality from cancer of the pharynx. All deaths attributed to cancer at this site occurred after 20 YSFE and all in terminated fire fighters. Three of the 4 cases occurred in subjects with 35+ years of employment and 2 of these 3 cases were cases of cancer of the oropharynx, resulting in a statistically significant excess (SMR=1176). All four deaths were histologically confirmed cases of pharyngeal cancer (two oropharynx and two hypopharynx), according to the OCTRF.

Cancer of the rectum and rectosigmoid junction (SMR=171)
Also suggestive of excess risk is death certified to this site. No death occurred before 10 YSFE, and there is little evidence of a

pattern according to YSFE. Cancer of the rectum increases with increasing age. All deaths occurred in subjects with 15 or more years of employment, and the SMRs are elevated for the 15-29 and 30+ duration categories. Of the 13 deaths, according to the OCTRF, 10 were incident cases of cancer of the rectum, while 3 were colon cancer cases. All were histologically confirmed, excluding two rectum cancer cases.

Cancer of the pancreas (SMR=140)
Of the 14 deaths, none occurred before 10 YSFE, and there is no apparent trend by calendar period or YSFE, although the overall SMR is somewhat elevated. Risk increases with increasing age. Deaths occur only in those with 10 years or more of employment.

Cancer of the bronchus and lung (SMR=95)
Although there is no evidence of increased risk for lung cancer in this study, this site is covered in the Howe and Burch review (1990). There is little evidence of consistently increasing SMRs as YSFE increases. There was no death before 10 YSFE or after 60 YSFE. Likewise, no pattern is apparent according to calendar period. Whereas about 7 deaths are expected before age 50, deaths from this cause occur only after age 50, but there is no pattern according to age among those over 50. Risk is elevated for those in the shortest duration category and statistically significantly decreased among current workers (SMR=34). All 51 lung cancer deaths were verified as lung cancer incident cases, excepting two certified to other sites. Of the lung cancer cases, all but seven were histologically confirmed.

Eight studies combine to give an SMR of 93 (95% CI: 84-103). Not included in this calculation is a study by Hansen (1990) comparing the mortality of male fire fighters to civil servants and salaried employees classified as such from census information. This study found a statistically significant increase in lung cancer for fire fighters (SMR=317). Hansen also reports an excess for "non-pulmonary" cancer, but provides no detail of specific cause.

Cancer of the prostate (SMR=132)
None of the 16 deaths occurs before 20 YSFE, and excluding this period the SMR is 136. While there is no pattern according to age at risk or calendar period, there is a statistically significant SMR for 1980-1984 (SMR=303). According to the five year groupings of duration of employment, there is one statistically significant excess at 25-29 years (SMR=329). While one death occurs in a subject with 5-9 years of employment, all others occur in those with 25 years or more duration.

Cancer of the testis (SMR=246)
Although there are only three deaths from cancer of the testis and they occurred in three different calendar periods, it is important to note that all three occurred among current workers within 10 years of first employment, giving a statistically significant SMR of 500. Two of the three deaths occurred after 1963, and both were histologically confirmed incident cases at the same site.

Lymphosarcoma and reticulum cell sarcoma (SMR=150)
There is a suggestion of increased mortality risk for this cancer site. Three of the four deaths are lymphosarcomas (SMR=203). There is some evidence of increasing risk as YSFE increases and an excess is seen at 10-14 years of employment. The risk is approximately equal for current and terminated workers. Of the four deaths, three had cancer incidence records after 1963 and two of these were histologically confirmed.

Lymphatic leukaemia (SMR=190)
Despite only four deaths for this cause, an elevated SMR is apparent. All four deaths occurred since 1970 in subjects 60 years of age and older, with at least 30 YSFE, and at least 30 years of fire department service. Records were available from the OCTRF for three of the four deaths, and all were incident cases of lymphatic leukaemia; however, only one was histologically confirmed.

Respiratory System Diseases

Chronic bronchitis, emphysema and asthma (SMR=130)
These diseases are usually lumped together under the term, Chronic Obstructive Pulmonary Disease or COPD. This group includes any disorder which is marked by persistent slowing of bronchial air flow (Higgins and Thom, 1988). There is some disagreement over whether or not asthma should be included in the definition, and in this study the one death from asthma is included.

After more than 20 YSFE, SMRs increase consistently with increasing 10 year periods of YSFE: 124, 137, 161 and finally 250 (not shown), indicating a diminishing healthy worker effect.

After more than 20 YSFE, SMRs increase consistently with increasing 10 year periods of YSFE: 124, 137, 161 and finally 250 (not shown), indicating a diminishing healthy worker effect. In the most recent calendar period, there is almost a statistically significant increase (SMR=206) for COPD. While there is low risk at less than 60 years of age, the SMR for those 60 years and older is significantly increased. SMRs vary substantially over the range of employment duration. The only death attributed to asthma was that of a man who had worked 35 years or more.

Non-occupational risk factors include smoking and air pollution. Some researchers contend that occupational factors include tolu-

ene diisocyanate, grain, wood and hardrock dust, and several irritant gases (Becklake et al., 1988). While chronic changes in pulmonary function have been documented in fire fighters (Sparrow et al., 1982; Peters et al., 1974), as has acute airway responsiveness (Sherman et al., 1989), many investigators contend that this is not reflected in cohort studies of mortality because risk is masked by the healthy worker effect (Becklake et al., 1988; Rosenstock et al., 1990). In comparison to police officers, one study has shown that fire fighters experience an excess risk of non-malignant respiratory diseases (SMR=141, Rosenstock et al., 1990). In the study of fire fighters in Edmonton and Calgary, COPD was moderately increased (SMR=157), but showed no relation to duration of employment (Guidotti, 1992).

Digestive System Diseases

Cirrhosis of the liver (SMR=133)
Although cirrhosis of the liver exhibits no excess overall, there is a statistically significant SMR during 1980-1984. Of the 26 cirrhosis deaths, no death occurred before 10 YSFE, and risk is highest after 20 YSFE. The SMRs are similar for the two broad age categories, but there is a statistically significant excess at ages 40-49. Excess risk is seen only for those working for 15-29 years. There is elevated risk only among terminated workers (SMR=207).

Digestive system diseases as a whole were elevated in San Francisco fire fighters due to excesses of cirrhosis and diseases of the stomach and duodenum, and the authors did not know whether to attribute this excess to fire fighting exposures, alcohol or their interaction (Beaumont, 1991).

Gallbladder diseases (SMR=296)
Gallbladder diseases account for only 5 deaths, but the overall SMR is elevated at almost 300. There is one statistically significant excess for the calendar period 1955-1959. All five deaths occurred at 30+ YSFE, at 60 years or older, and at 30+ years of employment.

Gallbladder diseases include cholelithiasis (gallstones), cholecystitis (inflammation of the gallbladder), and haematobilia (bleeding into the biliary passages). Known risk factors include obesity, trauma and a diet high in cholesterol (Schoenfield, 1977). We are not aware of any occupational risk factors that have been identified for mortality from these diseases, and results for this cause of death have not been reported in other studies of fire fighters.

Summary of Findings

Although there was no apparent excess risk due to mortality from cardiovascular diseases as a group in this cohort of fire fighters, for one specific cause of death in this group, aortic aneurysm, a statistically significant excess was demonstrated. Further, risk of death from acute myocardial infarction and diseases of the veins, lymphatics and other circulatory system diseases was somewhat elevated, particularly when one considers that these fire fighters were much healthier than the general population when they entered their profession.

In terms of cancer, statistically significant excesses were seen for cancer of the brain and other nervous system tumours and for "other" malignant neoplasms. Other cancer sites exhibiting elevated, although not statistically significant, risks were: pharynx, rectum and rectosigmoid junction, pancreas, prostate, lymphosarcoma and reticulum cell sarcoma, and lymphatic leukaemia. Some degree of elevated mortality risk was also seen for respiratory and digestive system diseases as follows: chronic bronchitis, emphysema, and asthma as a group, cirrhosis of the liver, and gallbladder diseases.

Discussion

This epidemiologic study suggests that fire fighting is a hazardous occupation in terms of increasing risk of death due to several causes, such as cancer of the brain and other tumours of the nervous system, "other" malignant neoplasms, and aortic aneurysm. There are also suggestive increases in risk for several other causes of death, including various cancers, and diseases of the respiratory, circulatory and digestive system.

Study limitations

As in any epidemiologic study, the associations reported here may be due to chance, bias, or the effects of factors not measured, such as smoking, alcohol or diet. In terms of smoking, since there is no elevation in risk for lung cancer, excess smoking by fire fighters compared to the general population is most likely not an explanation for any of the risks seen in this study.

Causal relationships are rarely established in one study. In addition to the strength of an association, it is biological plausibility and consistency across several studies that will establish a causal link between an occupation and a disease. It is also important to note that an epidemiologic study cannot definitively establish the absence of a risk but can often place an upper bound on overall risk. Furthermore, this study examined mortality. The extent to which this is an indicator of morbidity depends on the specific cause of death.

This study shares a common feature of occupational cohort studies in that no information is available on an individual's exposure. In the absence of information on individual fire fighter activity and fire hall assignment and in light of the small variation in job assignment of fire department employees, the only feasible index of exposure for this cohort study is number of years of employment. In this respect, the results are directly comparable to those from other studies.

Potential exposures

There is very little documentation of the prevalence of actual exposures which fire fighters receive. In an effort to obtain information specific to urban Canadian fire fighters, a group of researchers was commissioned to provide exposure data on fire fighters as part of their on-going study of cancer in Metropolitan Montreal (Siemiatycki, 1991). Exposure assessment is done by a team of industrial hygienists and chemists based on a detailed personal interview. Forty-seven fire fighters were interviewed, 40 of whom were from urban areas. Several substances were identified as exposures for the majority of fire fighters (85%-98% prevalence of exposure): polycyclic aromatic hydrocarbons (PAHs), pyrolysis fumes, aliphatic aldehydes, carbon monoxide, formaldehyde, particulates from wood and soot, nitrogen oxides, sulphuric acid, chlorinated alkanes, benzopyrene, chrysotile asbestos, sulphur dioxide, monocyclic aromatic hydrocarbons, chlorine, phosgene, engine emissions, carbon tetrachloride, solvents, alkanes, hydrogen cyanide, hydrogen chloride, plastics pyrolysis products, benzene, cyanide, and lubricating oils. It is known, however, that very different exposures can occur for two fire fighters at the same fire even if they are side by side (Brandt-Rauf et al., 1988). In experimental studies, carbon monoxide, formaldehyde and acrolein are among chemicals identified as pyrolysis products which occur even at relatively low temperatures (i.e., in the overhaul stage of a fire) (Edgerley, 1980).

In a recent study which measured substances in 22 fires (15 residential, 6 training, and 1 car fire) which occurred in various urban fire departments across the U.S., carbon monoxide was the most common contaminant (Jankovic et al., 1991). Distinctions were made between measurements taken in the knockdown (bringing the main body of fire under control) and overhaul (searching for and extinguishing hidden fires), and those taken inside and outside self-contained breathing apparatus. Ranked in order of decreasing occurrence in the knockdown phase, the substances found were: carbon dioxide, carbon monoxide, ethylene, methanol, methane, acetaldehyde, acrolein, benzene, hydrogen cyanide, and hydrochloric acid. Several of these chemicals were also measured, although at lower concentrations, in the overhaul phase, which is necessary mainly for larger fires. These investigators found that masks are usually not worn during the

overhaul phase and that the low concentrations found which were attributed to "inside mask" were due either to early removal or non-use of masks and not to leakage of the masks themselves. The increased use of synthetic materials since the 1950's has increased concern about fire fighters' exposure to potentially toxic chemicals.

Conclusion

This epidemiologic study suggests that fire fighting is a hazardous occupation at least in terms of increasing risk of death due to several causes, such as cancer of the brain and other tumours of the central nervous system, "other" malignant neoplasms, and aortic aneurysm. There are also suggestive increases in risk for several other causes of death, including various cancers, and diseases of the respiratory, circulatory and digestive systems. For several causes of death we have argued that, because fire fighters are selected for their excellent health, where moderate increases are apparent compared to the general population, these are probably underestimates of the magnitude of the true hazards of fire fighting.

TABLE 4
Standardized Mortality Ratios by Years Since First Employment for Selected Causes

	Years since first exposure								
	< 20 Years			*20-29 Years*			*≥ 30 Years*		
Cause	*Obs.*	*SMR*	*95% CI*	*Obs.*	*SMR*	*95% CI*	*Obs*	*SMR*	*95% CI*
All Causes	120	79	65-94	143	95	80-112	514	99	91-108
All Cancers	28	120	77-170	36	94	66-131	135	106	89-125
pharynx	0	0	—	1	122	3-679	3	188	39-549
rectum	1	125	3-696	2	146	18-527	10	179	86-329
pancreas	1	100	3-557	2	95	12-344	11	157	78-281
lung	1	23	1-128	13	103	55-176	40	100	71-136
melanoma	1	91	2-507	1	130	3-724	0	0	—
prostate	0	0	—	2	244	30-881	14	126	69-211
testis	3	333	69-973	0	0	—	0	0	—
brain & other nervous system	6	286	105-623	2	99	12-356	6	222	81-483
lymphatic leukaemia	0	0	—	0	0	—	4	267	73-684
other malignant neoplasms	1	143	4-797	4	288	78-737	15	306	171-505
Circulatory System	37	93	65-128	77	118	93-147	267	96	85-108
acute myocardial infarction	22	102	64-154	48	126	93-167	135	102	86-121
arteriosclerosis	1	100	3-557	3	165	34-482	20	141	86-218
aortic aneurysm	0	0	—	3	303	62-886	16	232	133-377
diseases of veins, lymphatics, etc.	1	143	4-797	2	211	25-760	5	161	52-376
Chronic bronchitis, asthma & emphysema	0	0	—	2	80	10-290	26	146	95-214
Cirrhosis of the liver	4	87	24-223	10	152	73-280	12	141	73-246
Gallbladder diseases	0	0	—	0	0	—	5	385	125-898

TABLE 5
Standardized Mortality Ratios by Age for Selected Causes

Cause	Age					
	< 60 Years			≤ 60 Years		
	Obs.	SMR	95% CI	Obs	SMR	95% CI
All Causes	337	91	81-101	440	97	88-107
All Cancers	87	107	86-132	112	104	86-125
pharynx	1	61	2-340	3	240	49-701
rectum	4	138	38-353	9	191	87-363
pancreas	4	97	26-248	10	170	81-313
lung	22	91	57-138	32	97	66-137
melanoma	2	93	11-336	0	0	—
prostate	2	153	19-553	14	130	71-218
testis	3	268	55-783	0	0	—
brain & other nervous system	10	198	95-364	4	204	56-522
lymphatic leukaemia	0	0	—	4	336	92-860
other malignant	9	304	139-577	11	266	133-476
Circulatory System	150	110	93-129	231	93	81-106
acute myocardial infarction	93	117	94-146	112	100	82-120
arteriosclerosis	4	113	31-289	20	149	91-230
aortic aneurysm	3	163	33-473	16	245	140-398
veins, lymphatics & other	3	148	31-433	5	181	59-422
Respiratory System	7	57	23-117	38	112	79-154
chronic bronchitis, asthma & emphysema	2	43	5-155	26	155	101-227
Digestive System	20	96	58-148	24	156	100-232
cirrhosis of the liver	18	130	77-205	8	138	60-272
gallbladder diseases	0	0	—	5	420	136-980

TABLE 6

Standardized Mortality Ratios by Duration of Employment for Selected Causes

	Duration of employment								
	< 15 Years			*15-29 Years*			*≤ 30 Years*		
Cause	*Obs.*	*SMR*	*95% CI*	*Obs.*	*SMR*	*95% CI*	*Obs*	*SMR*	*95% CI*
All Causes	130	88	74-104	240	97	85-110	383	91	82-101
All Cancers	36	137	96-190	53	87	65-114	100	100	81-122
pharynx	1	227	6-1265	0	0	—	3	236	49-699
rectum	0	0	—	5	220	71-513	8	180	78-355
pancreas	2	172	21-621	3	92	19-269	9	165	75-313
lung	8	129	56-254	16	83	47-135	27	87	57-127
melanoma	1	109	3-607	1	89	2-496	0	0	—
prostate	1	152	4-847	5	213	69-497	9	101	46-192
testis	3	353	73-1032	0	0	—	0	0	—
brain & other nervous system	5	258	84-602	3	105	22-307	5	233	76-544
lymphatic leukaemia	0	0	—	0	0	—	4	364	99-932
other malignant	4	426	116-1091	3	134	28-392	13	340	181-581
Circulatory System	40	97	69-132	126	113	94-135	207	91	79-104
acute myocardial infarction	23	108	68-162	73	120	94-151	105	98	80-119
arteriosclerosis	2	145	18-254	5	128	42-299	17	148	86-237
aortic aneurysm	1	143	4-797	5	242	79-565	13	239	127-409
Respiratory System	4	80	22-205	7	62	25-128	32	109	75-154
chronic bronchitis, asthma & emphysema	3	175	36-511	4	76	21-195	19	133	80-208
Digestive System	4	59	16-151	19	135	82-256	21	134	83-205
cirrhosis of the liver	4	100	27-256	17	189	110-303	5	78	25-182
gallbladder diseases	0	0	—	0	0	—	5	472	153-1101

References

Bates, J.T. Coronary artery disease in the Toronto fire department. Journal of occupational medicine. Vol.29,no.2 (Feb., 1987). p.132-135.

Beaumont, J.J.; Chu, G.S.T.; Jones, J.R.; Schenker, M.B.; Singleton, J.A.; Piantanida, L.G.; Reiterman, M. An epidemiologic study of cancer and other causes of mortality in San Francisco firefighters. American journal of industrial medicine. Vol. 19 (1991). p.357-372.

Becklake, M.R.; Bourbeau, J.; Menzies, R.; Ernst, P. The relationship between acute and chronic airway responses to occupational exposures. Current pulmonology. Vol 9 (1988). p.25-66.

Breslow, N.E.; Day, N.E. Statistical Analysis in Cancer Research, V. II: The Design and Analysis of Cohort Studies. Lyon: IARC Scientific Publications No. 82, 1987.

Demers, P.A.; Heyer, N.J.; Rosenstock L. Mortality among firefighters from three northwestern United States cities. British journal of industrial medicine. Vol.49 (1992) (in press).

Edgerley, J. Oxidative pyrolysis of plastics. Fire and materials. Vol.4,no.2(1980). p.77-82.

Eliopulos, E.; Armstrong, B.K.; Spickett, J.T.; Heyworth F. Mortality of fire fighters in western Australia. British journal of industrial medicine. Vol.41,no.2(May, 1984). p.183-187.

Grimes, G.; Hirsch, D.; Borgeson, D. Risk of death among Honolulu fire fighters. Hawaii medical journal. Vol. 50, no. 3 (1991). p.82-85.

Guidotti, T.L. Mortality of urban firefighters in Alberta, 1927-1987. American journal of industrial medicine (1992) (in press).

Hansen, E.S. A cohort study on the mortality of firefighters. British journal of industrial medicine. Vol. 47. (1990). p.805-809.

Heyer, N.; Weiss N.S.; Demers, P.; Rosenstock L. Cohort mortality of Seattle fire fighters: 1945-1983. American journal of industrial medicine. Vol.17 (1990). p.493-504.

Higgins, M.; Thom, T. Epidemiology and natural history of COPD. Current pulmonology. Vol.9 (1988). p.1-24.

Howe, G.R.; Burch, J.D. Fire fighters and risk of cancer: an assessment and overview of the epidemiologic evidence. American journal of epidemiology. Vol. 132, no. 6 (1990). p.1039-1050.

Jankovic, J.; Jones, W.; Burkhart, J.; Noonan, G. Environmental study of firefighters. Annals of occupational hygiene. Vol.35 (1991). p.581-602.

Kristensen, T.S. Cardiovascular diseases and the work environment. A critical review of the epidemiologic literature on chemical factors. Scandinavian journal of work, environment and health. Vol. 15 (1989a). p.245-264.

Kristensen, T.S. Cardiovascular diseases and the work environment. A critical review of the epidemiologic literature on nonchemical factors. Scandinavian journal of work, environment and health. Vol. 15 (1989b). p.245-264.

Lindsay, J.; Hurst, J.W. The Aorta. New York: Grune and Stratton, Inc., (1979).

Mastromatteo, E. Mortality in city firemen— II. A study of mortality in firemen of a city fire department. A.M.A. archives of industrial health. Vol.20 (Sept., 1959). p.227-233.

Meijers, J.M.M.; Swaen, G.M.H.; Volovics, A.; Lucas, L.J.; Van Vliet, K. Occupational cohort studies: The influence of design characteristics on the healthy worker effect. International journal of epidemiology. Vol.18,no.4 (1989). p.970-975.

Musk, A.W.; Monson, R.R.; Peters, J.M.; Peters, R.K. Mortality among Boston fire fighters, 1915-1975. British journal of industrial medicine. Vol.35,no.2 (May, 1978). p.104-108.

Peters, J.; Theriault, G.; Fine, L.; Wegman, D. Chronic effects of fire fighting on pulmonary function. New england journal of medicine. Vol.291 (1974). p.1320-1322.

Robins, J.M. A graphical approach to the identification and estimation of causal parameters in mortality studies with sustained exposure periods. Journal of chronic diseases. Vol.40(Suppl.2) (1987). p.139S-161S.

Rosenstock, L.; Demers, P.; Heyer, N.J.; Barnhart, S. Respiratory mortality among fire fighters. British journal of industrial medicine. Vol.47 (1990). p.462-465.

Sama, S.R.; Martin, T.R.; Davis, L.K.; Kriebel, D. Cancer incidence among Massachusetts firefighters, 1982-1986. American journal of industrial medicine. Vol. 18 (1990). p.47-54.

Shannon, H.; Jamieson, E.; Walsh, C.; Julian, J.; Fair, M.; Buffet, A. Comparison of individual follow-up and computerized record linkage using the Canadian National Mortality Data Base. Canadian journal of public health. Vol.80 (1989). p.54-57.

Sherman, C.B.; Barnhart, S.; Miller, M.F.; Segal, M.R.; Aitken, M.; Schoene, R.; Daniell, W.; Rosenstock, L. Firefighting acutely increases airway responsiveness. American review of respiratory disease. Vol.140 (1989). p.185-190.

Shoenfield, L.J. Diseases of the gallbladder and biliary system. New York: John Wiley and Sons (1977).

Siemiatycki J. Risk factors for cancer in the workplace. Boca Raton: CRC Press (1991).

Sinks, T.; Smith, A.B.; Rinsky, R.; Watkins, K.; Shults, R.; Steele, G. Health Hazard Evaluation Report HETA-89-116-2094 Westing-house Electric Corporation, Bloomington, Indiana. Cincinnati, Ohio: NIOSH (Jan., 1991).

Sparrow, D.; Bosse, R.; Rosner, B.; Weils, S. The effect of occupational exposures on pulmonary function— a longitudinal evaluation of fire fighters and non-firefighters. American review of respiratory diseases. Vol.123 (1982). p.319-322.

Thomas, T.L.; Waxweiler, R.J. Brain tumours and occupational risk factors: A review. Scandinavian journal of work, environment, and health. Vol.12 (1986). p.1-15.

Tornling, G.; Gustavsson, A.; Gustavsson, P.; Hogstedt, C.; Soder-holm, M. Dodsorsaker bland brandman i stockholm. Omradesvisa undersokningar av kemiska miljoer (25: Dec., 1990).

Vena, J.E.; Fiedler, R.C. Mortality of a municipal-worker cohort; IV. fire fighters. American journal of industrial medicine. Vol.11, no.6 (1987). p.671-684.

Scientific Report: Mortality Study of Fire Fighters in Metropolitan Toronto

Kristan A. L'Abbé and George A. Tomlinson

Industrial
Disease
Standards
Panel

Scientific Report: Mortality Study of Fire Fighters in Metropolitan Toronto

Kristan A. L'Abbé and George A. Tomlinson

Industrial
Disease
Standards
Panel

SCIENTIFIC REPORT: MORTALITY STUDY OF FIRE FIGHTERS IN METROPOLITAN TORONTO

Kristan A. L'Abbé and George A. Tomlinson

March 17, 1992

"We don't know exactly what that mix of chemicals produces, but we do know it's something you don't want to breathe." - Grant Nicurity, co-ordinator of disaster services in Regina, discussing a fire at a chemical storage facility in Grand Coulee, Saskatchewan, as quoted in The Globe and Mail, Nov. 21, 1991.

Acknowledgements: We wish to thank all fire department employees who were enroled in this study, and we are grateful for the cooperation of unions and management in this effort. In particular, we thank Chief Joe Gibson, Dr. J.T. Bates, Evelyn Funston, and Jim Pullen. The protocol was initiated by Dr. Lesbia Smith. The data collection phase was greatly assisted by four enthusiastic research assistants, Wendy Findlay, Jani Gendron, Jodi Gendron, and Debbie Morin. We also acknowledge the contributions of Statistics Canada (Martha Fair, Maureen Carpenter, and Pierre Lalonde), the provincial and territorial Registrars of Vital Statistics, the Ontario Cancer Treatment and Research Foundation (Eric Holowaty and Darlene Dale), peer reviewers, and the staff of the Industrial Disease Standards Panel.

Canadian Cataloguing in Publication Data

L'Abbé, Kristan A.
Scientific report of the mortality study of fire fighters in Metropolitan Toronto

(IDSP occasional paper)
Includes bibliographical references.
ISBN 0-7729-9793-4

1. Fire fighters–Ontario–Toronto Metropolitan Area–Mortality. I. Tomlinson, George, 1964- . II. Ontario. Industrial Disease Standards Panel. III. Title. IV. Series.

HB1323.P82C35 1992 304.6'4'0883633 C92-092553-7

SCIENTIFIC REPORT: MORTALITY STUDY OF FIRE FIGHTERS IN METROPOLITAN TORONTO

TABLE OF CONTENTS

N.B., Page # refers to bottom centre number.

		PAGE
1.	INTRODUCTION	1
2.	METHODS	2
3.	RESULTS	5
4.	DISCUSSION	17
	Study Limitations	17
	Potential Exposures	20
5.	CONCLUSION	26
6.	REFERENCES	28
7.	LIST OF TABLES	32
8.	LIST OF APPENDICES	48
	Appendix A - Protocol	A-1 to A-29
	Appendix B - Code Conversions	B-1 to B-13
	Appendix C - Descriptive Tables	C-1 to C-5
	Appendix D - Memo on Index of Exposure	D-1 to D-5
	Appendix E - Occupational Exposures	E-1 to E-11

SCIENTIFIC REPORT:
MORTALITY STUDY OF FIRE FIGHTERS IN METROPOLITAN TORONTO

1. INTRODUCTION

The Industrial Disease Standards Panel (IDSP) initiated a study of mortality among fire fighters after receiving a submission from a union that expressed concern about cardiovascular disease among its fire fighters. There is conflicting evidence of increased risk of cardiovascular disease among fire fighters. Two previous studies of mortality among fire fighters in the City of Toronto have shown slight excesses in cardiovascular mortality (Mastromatteo, 1959; Bates, 1987). One recent proportional mortality study of fire fighters in Hawaii showed a borderline elevation for death from cardiovascular diseases (Grimes et al., 1991). However, other studies carried out in Australia, Canada and the United States have not found an overall increase in cardiovascular mortality (Musk et al., 1978; Eliopulos et al., 1984; Vena and Fiedler, 1987; Beaumont et al., 1991; Demers et al., 1992; Guidotti, 1992). But within some of these studies, sub-groups with either long duration of employment or time since first exposure have exhibited excess risk (Vena and Fiedler, 1987; Demers et al., 1992).

Several studies of fire fighters have demonstrated increased risk for cancer at various sites. A report that combines the results from published studies suggests that fire fighters experience excess mortality from brain cancer and possibly from multiple myeloma (Howe and Burch, 1990).

The protocol for a retrospective cohort study of fire fighters was finalized in January, 1990 (Appendix A), following peer review. Data collection began shortly thereafter and was completed in June, 1990. Following a cleanup of the data and an effort to obtain missing information from fire departments, the records were submitted to Statistics Canada in August, 1990 for computerized record linkage. These data were returned in September, 1991, with personal identifiers removed. This report presents the results of the study.

2. METHODS

The purpose of this study was to determine whether or not there is excess mortality among Metropolitan Toronto fire fighters, with particular attention being paid to cardiovascular disease. Excess mortality is defined as risk of death from any specific cause that is significantly above expectation as determined from the cause-specific mortality rates in a reference population. To some extent, excess mortality is an indicator of excess morbidity, depending on the specific cause of death.

A retrospective cohort study was conducted, with subjects being recruited from all six fire departments within Metropolitan Toronto: the Cities of Toronto, North York, Scarborough, Etobicoke and York and the Borough of East York. The inclusion of forces from six Metropolitan areas increases both the power of the study and the applicability of its results to urban, professional fire fighters.

A standard data collection instrument was developed for ease of entering information directly into a computer data base. Four data abstractors were briefed on issues of confidentiality and were required to take an oath of secrecy. Training in abstracting procedures was provided and coding practices were checked periodically for accuracy and interobserver reliability.

Records of employment histories were located in fire department headquarters, personnel offices and archives. The quality of these records varied with fire department, calendar time and the media used for record-keeping. All departments maintain paper or card files to some extent, some use microfilm or microfiche and recently some have been storing information on computer.

At present, all fire departments keep records of each fire fighter's name, sex, date of birth, date of hire, date of termination, reason for termination, current and past job titles, assignments (to a particular division), social insurance number and other identifying numbers, such as employee number. Older records are more likely to be incomplete and often lack information about job titles and fire hall assignments.

Several temporal variables were calculated to supplement those mentioned above. These included age at hire, age at exit (death or end of follow-up) from the cohort, time since first employment and duration of employment.

In the past, fire department employees were not generally assigned to separate divisions. A fire fighter may have performed a distinct duty, and would perhaps have been reassigned if he suffered health problems. Unfortunately, little, if any, of this sort of information is to be found in older personnel files. More recently, fire department employees have been assigned to one of six divisions: 1) administration; 2) fire prevention; 3) training; 4) repairs and maintenance; 5) communications; and 6) fire fighting. Our understanding from discussions with fire chiefs is that fire department employees have recently tended to keep their divisional assignments throughout their careers. Exceptions to this tendency are some senior administrators, such as Deputy Chiefs and Chiefs, who started out as fire fighters. Furthermore, recent job changes have been well-documented in personnel files. However, because of the sparsity of fire division assignment information from the past, we cannot be sure that employees who did not have fire fighting listed as their division were never fire fighters. Communications with fire chiefs lead us to believe that most of them at one time or another were in fact fire fighters. As a result, the few employees with assignments to divisions other than fire fighting were included in the analysis. They comprise 13 deaths.

A data file was created containing information on each fire fighter for whom fire department records could be located. This file included personal identifiers as well as other variables of interest such as marital status and fire department. Personal identifiers include first name, middle initial, last name, date of birth, sex and social insurance number. The inclusion of identifiers additional to name and date of birth increases the probability of correct matching by Statistics Canada.

This data file was sent to Statistics Canada (SC). Through record linkage with the Canadian Mortality Data Base (CMDB) (Smith and Newcombe, 1982), SC determined the date and cause of death for any cohort members who were recorded as having died between 1940 and 1989. The CMDB contains death certificate information for all occurrences of death in Canada (and limited information on death of Canadian residents outside of Canada) between 1940 and the present, as reported to SC by the Registrars of Vital Statistics for each province and the territories. The CMDB computerized record linkage procedure (CRL) is a reliable means of determining occurrences of death and cause of death when it is provided with the full name and date of birth of an individual (Shannon et al., 1989).

Once the matching process was complete, SC added to the data file the date and cause of death for each cohort member who

was determined to be dead. The cause of death was provided both in the International Classification of Disease (ICD) revision in use at the time of death and in the 9th ICD revision, the conversion being made where necessary by experienced nosologists at SC. A table of equivalencies between ICD revisions is provided in Appendix B. SC provided rates for Ontario males for causes of death identified by Laboratory Centre for Disease Control (LCDC) codes. The LCDC codes group ICD 9th revision codes for related causes of death and are occasionally not mutually exclusive. This can be seen in the conversion table in Appendix B showing LCDC and ICD code equivalencies. That is to say, someone who died from one ICD9 cause of death could be classified as dying from two different LCDC codes, as a few LCDC categories contain the same ICD codes. Results in this report are presented in terms of LCDC codes and in the Tables an effort has been made to present categories of cause of death which are mutually exclusive. Sub-groups of overall causes of death are represented by indentations in the Tables.

SC then removed all personal identifiers from this file and returned it to the IDSP staff for statistical analysis. Cohort members not identified as dead through the CMDB record linkage procedure were assumed to be alive, in accordance with what other researchers have done. This is reasonable in that the number of observed deaths and number of expected deaths are then based on the same method of death ascertainment carried out by SC. Although there was clear documentation in fire department records of thirteen additional deaths that were not found in the CMDB record linkage procedure, these were not included in the analysis. Based on this figure, an estimate of the minimum percentage of deaths missed by SC is 1.7% (13/765). The maximum percentage of deaths missed by SC has elsewhere been estimated at about 4% (Shannon et al., 1989).

For various reasons, not all fire fighters were eligible for inclusion in the final analysis. The small number of females in the cohort necessitated their exclusion. Because there is a training period of approximately six months for recruits, fire fighters who had worked for less than six consecutive months were excluded. Although mortality of the cohort was ascertained from 1940 through December 31, 1989, reference rates are available only for 1950 onward. Therefore, the mortality analysis begins January 1, 1950 and fire fighters who were not known to be alive on or after this date were excluded. Other requirements of the analysis meant that fire fighters without a known birth or hire date were excluded. Fire fighters who were not currently employed but who had no known termination date were excluded from all analyses involving duration of employment.

Mortality of the cohort specific to age, calendar period, and cause was compared to that of the Ontario male population over the period 1950-1989. To accomplish this, stratum-specific person-years were calculated using a modified life-table approach and the Person-years software package (Coleman et al, 1989). Subjects began to contribute person-years at risk on January 1, 1950 or their first day of employment, whichever was later and ceased to contribute person-years on their date of death, upon reaching age 85 or on December 31, 1989, whichever was earlier. The protocol called for censoring at age 85 on the grounds that certification of cause of death in elderly people is less reliable. The number of deaths expected from a particular cause was computed by multiplying the person-years in each age- and calendar period-specific stratum by the cause-specific Ontario mortality rates for that stratum. The ratio of the number of deaths observed to the number of deaths expected, when multiplied by 100, yields the standardized mortality ratio (SMR). Thus, an SMR greater than 100 may indicate an excess risk of death due to a particular cause. The statistical significance of an apparent excess is judged using a 95% confidence interval (implicitly, an alpha level of 0.05), assuming an underlying Poisson distribution of the number of observed deaths (Breslow and Day, 1987).

Information on deaths which were certified to any type of cancer was sent to the Ontario Cancer Treatment and Research Foundation (OCTRF). From records which date back as far as 1964, they returned confirmation of the cause of death and information on cancer incidence, including histological type and date of diagnosis. This may be particulary important, for example, in determining whether a brain cancer is a primary or secondary tumour. If it is secondary, it should not appear as the primary cause of death on the death certificate. However, misclassification of this kind is not uncommon, as it is often difficult in practice to distinguish between primary and secondary tumours, particularly when time has elapsed between the two or there has not been continuity of health care.

3. RESULTS

Although the anticipated size of the cohort was 4100, information on 5995 individuals was collected. Once record linkage had been completed, it turned out that 28 subjects had duplicate entries in the data file as a result of working for two fire departments or in two different periods. Further exclusions included 146 women, 65 men who were known dead before 1950, 58 men who were not known alive

in or after 1950, 48 men with unknown birth date, 24 men who had no date of hire, and 211 men who worked for less than six months. There were 41 men who were not current employees and who had no known termination date and who therefore could not be included in any analyses involving duration of employment. There was one additional exclusion of a man who reached age 85 before 1950 and who was therefore censored before the study began.

For the 5414 fire department employees included in the final analysis, descriptive statistics are seen in Table 2. The average age at hire was 25 years (standard deviation (sd)=3.8), while the mean interval from first employment to the end of follow-up was 24 years (sd=14.6). The average duration of employment for the 5373 subjects with complete information was 20 years (sd=12), while the average length of follow-up was 21 years (sd=11.8), ranging from 19 to 23 in the six Metropolitan Toronto fire departments. The distribution of follow-up is shown in Appendix C; 57% of the cohort have been followed for 20 years or more (including 29% who have been followed for 30 years or more). The total number of person-years at risk accumulated by this cohort is 116 201, and the distribution according to calendar year and years since first employment is seen in Table 3. Additional descriptive tables can be found in Appendix C.

At the end of follow-up (the earlier of date of death or Dec. 31, 1989), 55% (2931/5373) of the subjects whose employment status could be determined were classified as current, while the remaining 45% (2442/5373) were retired or terminated. The majority of subjects (65%) who retired or quit did so between age 50 and 70 years. Of the total number of fire department employees, 5251 (97%) were classified as fire fighters while 163 subjects were assigned to other fire department divisions.

From January 1, 1950 through Dec. 31, 1989, there were 837 deaths in the cohort, and the mean age at death was 63 years (sd=15), with the average ranging from 45 to 65.5 years in the six fire departments, while the minimum and maximum ages at death were, respectively, 22 and 95. As 60 deaths occurred at age 85 or after (and were censored), the analysis included 777 deaths (14% of 5414) under age 85, and 763 (98%) of these were among fire fighters. Of the 777 deaths, 229 (29%) died while still on the fire department payroll records or within one month of termination, while 473 (61%) died more than two years following date of termination, with the remainder (10%) dying between these two intervals.

Cause-specific mortality for the total cohort (n=5414) is presented in Table 5. The all-cause SMR is 94 (95%

confidence interval (CI): 88-101); therefore, no "healthy worker effect" is apparent overall. Most of the major groups of causes of death have SMRs of about 100; these include circulatory system diseases (384 deaths) with an SMR of 99 (95% CI: 89-109) and cancer (199 deaths) with an SMR of 105 (95% CI: 91-120). The two exceptions among the major groups of causes of death are infective and parasitic diseases (1 death, SMR=14, 95% CI: 0.4-77) and endocrine, nutritional and metabolic diseases (5 deaths, SMR=34, 95% CI: 11-80) which exhibit statistically significant deficits in mortality.

Among specific causes of death, two causes exhibit statistically significant deficits in mortality:
(1) diabetes mellitus (ICD9 250), 4 deaths, SMR=34, 95% CI: 9-88; and,
(2) chronic rheumatic heart disease (ICD9 393-398, 424.0, 424.1, 424.3), 1 death, SMR=15, 95% CI: 0.4-85.

Statistically significant excesses are seen for three specific causes of death:
(1) cancer of the brain and other nervous system tumours (ICD9 191-192), 14 deaths, SMR=200, 95% CI: 109-336;
(2) "other" malignant neoplasms (ICD9 195-199), 20 deaths, SMR=282, 95% CI: 173-436; and,
(3) aortic aneurysm (ICD9 441.0-441.6), 19 deaths, SMR=226, 95% CI: 136-353.
These will be discussed extensively below.

Elevated, although not statistically significant, risks (judged here as SMRs over 130 with 4 or more deaths) are also apparent for several additional causes of death:
(1) cancer of the pharynx (ICD9 146-148,149), 4 deaths, SMR=139, 95% CI: 38-357;
(2) cancer of the rectum and rectosigmoid junction (ICD9 154), 13 deaths, SMR=171, 95% CI: 91-293;
(3) cancer of the pancreas (ICD9 157), 14 deaths, SMR=140, 95% CI: 77-235;
(4) cancer of the prostate (ICD9 185), 16 deaths, SMR=132, 95% CI: 76-215;
(5) lymphosarcoma and reticulum cell sarcoma (ICD9 200.0, 200.1), 4 deaths, SMR=150, 95% CI: 41-384, of which 3 are lymphosarcoma (SMR=203, 95% CI: 42-592);
(6) lymphatic leukaemia (ICD9 204), 4 deaths, SMR=190, 95% CI: 52-485;
(7) lymphatic and myeloid leukaemia (ICD9 204, 205), 8 deaths, SMR=143, 95% CI: 62-282;
(8) all leukaemia (ICD9 204-206,207.0,207.2,207.8,208), 10 deaths, SMR=133, 95% CI: 64-245;
(9) chronic bronchitis, emphysema, and asthma (ICD9 490-493, 496), 28 deaths, SMR=130, 95% CI: 87-189;
(10) arteriosclerosis (diseases of arteries, arterioles, and

capillaries) (ICD9 440-448), 24 deaths, SMR=141, 95% CI: 91-210;
(11) diseases of veins, lymphatics and other circulatory (ICD9 415.1, 451-459), 8 deaths, SMR=167, 95% CI: 72-330;
(12) cirrhosis of the liver (ICD9 571), 26 deaths, SMR=133, 95% CI: 87-195; and,
(13) gallbladder diseases (ICD9 574-576), 5 deaths, SMR=296, 95% CI: 96-690.

Results from the analyses by years since first employment (YSFE) and calendar time, age at risk, and duration of employment for selected causes of death are summarized in Tables 6, 7, and 8, respectively. In this study, duration of employment is the only measure of occupational exposure; this issue will be expanded upon in the Discussion section. The Pearson correlation coefficient for years since first exposure and duration of employment is 0.84 (probability of less than 0.01 that the correlation equals zero). Since the correlation between these variables is very high, the analyses by duration of employment and by years since first employment are not independent: they are measuring changes in risk along very similar dimensions.

The results according to YSFE, calendar period, age at risk and duration are presented below for all causes of death for which we have judged the findings to be interesting according to at least one of the following criteria: statistically significant excesses are seen; no excesses are seen when they may have been expected based on previous literature; or noteworthy patterns according to the temporal variables are apparent. There is essentially no variability within the cohort according to age at hire (92% of the cohort were hired between 20 and 29 years of age) and therefore analysis according to age at hire is not presented. Since 98% of deaths were among fire fighters, it is impossible to discern if differences exist according to job division and title; therefore, this analysis is not presented. Further, 52% of the cohort were enroled from the largest and longest established of the six fire departments, and 79% of the deaths were from this department; therefore, analysis by fire department within Metropolitan Toronto was not conducted. Although an analysis was also conducted according to whether the subject was a current worker or had terminated employment, results are discussed only if they shed additional light on the pattern of risk. If information was provided by the OCTRF for cancer deaths, this is presented. According to the confidentiality restrictions of the OCTRF, it is not possible to divulge detailed information regarding case groups of less than five. All-cause mortality is presented first, followed by causes which exhibit statistically significant increases overall and then several others (in order of their

occurrence in ICD9) which meet the above criteria.

All causes (SMR=94, 95% CI: 88-101)

All-cause mortality shows an increasing trend in SMRs as YSFE increase (Table 6), from 79 (95% CI: 65-94), to 95 (95% CI: 80-112), to 99 (95% CI: 91-108). In the more detailed analysis by 10 year categories of YSFE (not shown), the SMRs (95% CIs) are 66 (48-90), 88 (70-110), 95 (80-112), 92 (79-106), 104 (90-120), 101 (84-120) and 98 (56-158). Therefore, a strong healthy worker effect is evident for those with less than 10 YSFE, but not at greater than 10 YSFE.

For all calendar periods, the 95% CIs for the SMRs encompass 100, and the SMRs themselves range from 71 to 109. A strong healthy worker effect is evident for those at risk under age 40 (56 deaths, SMR=64, 95% CI: 48-83), while at 40-49 years of age (103 deaths, SMR=97, 95% CI: 80-118) and older, the 95% CIs encompass 100. In other words, by the time a fire fighter reaches the age of forty, he is no longer at a reduced risk of death compared to the general population. The SMR is 97 (95% CI: 88-107) at age 60 years or older whereas it is 91 (95% CI: 81-108) before this age (Table 7). According to years of employment (Table 8), the SMR rises from 88 among those with less than 15 years to 97 among those with 15 to 29 years and then falls slightly to 91 for those who worked 30 years or more. As expected, mortality risk among current workers is very low (190 deaths, SMR=56, 95% CI: 48-64), and that among terminated workers is high (563 deaths, SMR=119, 95% CI: 109-129).

Cancer of the brain and other nervous system (SMR=200, 95% CI: 109-336)

Cancer of the brain and other tumours of the nervous system were responsible for 14 deaths. An excess risk is seen in all calendar periods examined except 1975 through 1984 and the highest excess occurs in 1965-1969 (3 deaths, SMR=469, 95% CI: 97-1370). Large excesses are also seen for 0-19 YSFE and 40-59 YSFE, but there is no evidence of an excess for 20-39 YSFE, although this interpretation is based on small numbers. The level of risk is identical for relatively young (less than 60 years) (10 deaths, SMR=198, 95% CI: 95-364) and older fire fighters (4 deaths, SMR=204, 95% CI: 56-522) (Table 7). Among those with complete information for duration analysis, there were 13 deaths from brain cancer. Breakdown by five year groupings of duration of employment revealed that no deaths occurred in those with less than 5 years of employment but that the risk is statistically significant at 5 to 9 years duration (4

deaths, SMR=625, 95% CI: 170-1600, analysis not shown). According to the broad categories of duration (Table 8), risk exhibits a bimodal pattern, showing an excess for employees working less than 15 and more than thirty years, but not differing from that in the general population for the intervening period. However, confidence intervals overlap and the SMRs are relatively unstable due to small numbers.

Brain cancer became a specific cause of death in 1969 when the 8th revision of the International Classification of Diseases came into effect. When nosologists at Statistics Canada reviewed the 6 death certificates for deaths occurring before 1969, originally coded as ICD6 or ICD7 193 (nervous system tumours), all 6 were recoded as ICD9 191 (specifically brain cancer). However, the appropriate reference rates are not available for these cases (this would require that reference rates for those years be recoded into ICD9). The most cautious approach in the analysis is to recode these cases to ICD9 192, thereby using the broader cause of death category (cancer of the brain and other nervous system tumours, ICD9 191-192) for which reference rates are available, and this is what we have presented. As an alternative, the reference rates for brain cancer alone (ICD8 and ICD9 191) can be extrapolated back to 1950 (i.e., through ICD6) and these can be used to obtain the expected number of deaths from brain cancer alone. When this is done, an approximation of the SMR for brain cancer alone, given 14 observed deaths and 6.70 expected, is 209 (95% CI: 114-351). This SMR probably slightly underestimates the true SMR for brain cancer since the brain cancer mortality rate for males in Canada increased 28.5% between 1962 and 1984 (Chu, 1991). Extrapolation to the past of a higher rate would slightly overestimate the expected number of deaths, and thus underestimate the ratio of observed to expected.

According to information received from the OCTRF, one subject whose primary cause of death was recorded as another cancer site was recorded as an incident case of brain cancer in the Ontario Cancer Registry. The date of the brain cancer diagnosis was less than one year prior to the death. The certification of the death to another site of cancer can be thought of as an error, in that the other site was most likely secondary to the primary brain cancer (Dr. Holowaty, OCTRF, personal communication). The histological type was "glioblastoma, not otherwise specified."

The remaining nine subjects who died from brain cancer after 1963 were registered as incident brain cancer cases at the OCTRF and six of these were histologically confirmed, all as gliomas.

[Within the total cohort enumerated, there was one additional death certified as cancer of the brain and other nervous system, but the subject was excluded from the analysis based on the criterion that the death occurred before 1950.]

Other malignant neoplasms (SMR=282, 95% CI: 173-436)

"Other" malignant neoplasm ("ill-defined cancer") was certified for 20 deaths among fire fighters. There is some excess risk apparent in all calendar periods. No death occurred before 10 YSFE, and after this time the SMR increases as YSFE increases, until 50+ YSFE. The highest SMRs for this cause are seen for 40-49 YSFE (8 deaths, SMR=465, 95% CI: 201-916) and during 1975-1979 (6 deaths, SMR=588, 95% CI: 216-1280). According to age at risk, there are two periods of statistically significant excess: 7 deaths occurred at ages 50-59 (SMR=372, 95% CI: 150-767); and, 6 deaths occurred at ages 70-85 (SMR=311, 95% CI: 114-677). According to five year periods of employment, there are two statistically significant excesses, one at 10-14 years (3 deaths, SMR=750, 95% CI: 155-2192), and one at 35+ years (8 deaths, SMR=374, 95% CI: 161-737). Although one death occurs in a worker with 1-4 years of employment, all others attributed to this cause occur in those with 10 or more years of employment. By broader duration categories, these SMRs follow the same bimodal pattern as that for brain cancer, where risk is very high, then about the same as in the general population, and then quite high again. This cause of death is elevated among current (7 deaths, SMR=271, 95% CI: 109-559) and terminated workers (13 deaths, SMR=293, 95% CI: 156-502) alike.

Of these 20 "other malignant neoplasm" deaths, 17 were diagnosed with cancer after 1963. The OCTRF certified 11 of these subjects as "ill-defined site" incident cases. Of the remaining six subjects, two had cancer incidence of the same site and the other four had different sites.

Aortic aneurysm (SMR=226, 95% CI: 136-353)

For the 19 deaths from aortic aneurysm, the largest SMR is seen for the calendar period 1960-1964 (5 deaths, SMR=758, 95% CI: 246-1768). No death occurred before 20 YSFE, but at 20-30 YSFE the SMR rises to 303. At 40-49 YSFE, there is a statistically significant SMR of 295 (8 deaths, 95% CI: 127-582). Risk increases with increasing age, from 162 (95% CI: 33-473) at less than 60 years to a statistically significant risk of 245 (95% CI: 140-398) at 60 years of age and older (Table 7). Risk also increases from the first to second category of duration of employment (Table 8), then remains

stable. The majority of deaths (17/19) occurred in those who worked 25 years or more (SMR=252, 95% CI: 147-404). Although one death occurred in a fire fighter with 1-4 years of employment, all others occurred in those with 15 or more years of employment, and risk is elevated only for terminated workers (17 deaths, SMR=267, 95% CI: 155-427).

Cancer of the pharynx (SMR=139, 95% CI: 38-357)

There were four deaths attributed to cancer at this site, none occurring before 20 YSFE, and all in terminated fire fighters. Excluding the first 20 years since first employment, the SMR is 160 (95% CI:44-410). Three deaths occurred in men with 35 or more years of employment (SMR=476, 95% CI: 98-1392), while the remaining case had worked 5-9 years. Two of the 4 cases were actually cases of cancer of the oropharynx (ICD9 146) which occurred at 35 or more years of employment, resulting in a statistically significant SMR of 1176 (95% CI: 143-4250). All four deaths were histologically confirmed cases of pharyngeal cancer (two oropharynx and two hypopharynx), according to the OCTRF.

Cancer of the rectum and rectosigmoid junction (SMR=171, 95% CI: 91-293)

For the 13 deaths attributed to this cause, there is a statistically significant excess in the calendar period 1975-1979 (6 deaths, SMR=504, 95% CI: 185-1097). No death occurred before 10 YSFE, and there is little evidence of a pattern according to YSFE. Cancer of the rectum increases with increasing age (Table 7) from 138 (95% CI: 38-353) to 191 (95% CI: 87-363). All deaths occurred in subjects with 15 or more years of employment, and the SMRs are elevated for the 15-29 and 30+ duration categories (Table 8). Of the 13 deaths, according to the OCTRF, 10 were incident cases of cancer of the rectum, while 3 were colon cancer cases. All were histologically confirmed, excluding two rectum cancer cases.

Cancer of the pancreas (SMR=140, 95% CI: 77-235)

Of the 14 deaths, none occurred before 10 YSFE, and there is no apparent trend by calendar period or YSFE. Risk increases with increasing age, from an SMR of 97 (4 deaths, 95% CI: 26-248) for those less than 60 years of age, to 170 (10 deaths, 95% CI: 81-313) for older subjects. According to the broad divisions of employment duration, the SMRs exhibit a bimodal pattern, with increased risk in the longest and shortest duration categories and risk approximately the same as the general population in the intermediate category. However the confidence intervals do

overlap. Deaths occur only in those with 10 years or more of employment.

Cancer of the bronchus and lung (SMR=95, 95% CI: 71-123)

For the 54 deaths from this causes, there is little evidence of consistently increasing SMRs as YSFE increases, with SMRs of 27, 103, 97, 99, and 121 from 10-20 YSFE through 50-60 YSFE (not shown); there were no deaths before 10 YSFE or after 60 YSFE. According to more broadly defined categories of YSFE, the SMRs are 23 (95% CI: 1-126) at less than 20 YSFE, 103 (95% CI: 55-176) at 20-29 YSFE, and 100 (95% CI: 71-136) at 30+ YSFE. No pattern is apparent according to calendar period. Whereas about 7 deaths are expected before age 50 (SMR=0, 95% CI: 0-53), deaths from this cause occur only after age 50 (ages 50-59, 22 deaths, SMR=127, 95% CI: 80-193), and there is no pattern according to age (ages 60-69, 14 deaths, SMR=72, 95% CI: 39-120; and ages 70-85, 18 deaths, SMR=134, 95% CI: 79-211). Risk is elevated for those in shortest duration category (Table 8). Within this category, only those with 5-9 years employment clearly exhibit increased risk (5 deaths, SMR=256, 95% CI: 83-598). Within the second narrow category of duration, those with 20-24 years service also experience increased risk of lung cancer death (10 deaths, SMR=172, 95% CI: 82-317). However, risk is statistically significantly decreased among current workers (7 deaths, SMR=34, 95% CI: 14-70). All lung cancer deaths were verified as lung cancer incident cases, excepting two certified to other sites. Of the lung cancer cases, all but seven were histologically confirmed.

Cancer of the prostate (SMR=132, 95% CI: 76-215)

None of the I6 death occurs before 20 YSFE, and excluding this period the SMR is 136 (95% CI: 78-221). While there is no pattern according to age at risk or calendar period, there is a statistically significant SMR for 1980-1984 (6 deaths, SMR=303, 95% CI: 111-660). According to the five year groupings of duration of employment, there is one statistically significant excess at 25-29 years (5 deaths, SMR=329, 95% CI: 107-768). While one death occurs in a subject with 5-9 years of employment, all others occur in those with 25 years or more duration.

Cancer of the testis (SMR=246, 95% CI: 51-719)

Although there are only three deaths from cancer of the testis, it is important to note that all three occurred among current workers within 10 years of first employment, giving a statistically significant SMR of 500 (95% CI: 103-1461). The deaths occurred in three different calendar periods, but all three deaths occurred in fire fighters with

less than 10 years of employment, producing an SMR of 469 (95% CI: 97-1371). Two of the three deaths occurred after 1963, and both were histologically confirmed incident cases at the same site, according to the OCTRF.

Lymphosarcoma and reticulum cell sarcoma (SMR=150, 95% CI: 41-384)

Three of the four deaths are lymphosarcomas (SMR=203, 95% CI: 42-592). There is some evidence of increasing risk as YSFE increase, as well as a statistically significant excess at 10-14 years worked (SMR=833, 95% CI: 101-3010). The other two deaths in this category are in workers with 25 years or more of employment, and risk is approximately equal for current and terminated workers. Of the four deaths, three had cancer incidence records after 1963. One of the two lymphosarcomas was histologically verified as a cancer incidence case of lymphosarcoma. The other death certified as lymphosarcoma was a histologically confirmed case of reticulosarcoma and while the one death from reticulosarcoma was also recorded as a cancer incidence of reticulosarcoma, there was no record of histological confirmation.

Lymphatic leukaemia (SMR=190, 95% CI: 52-485)

All four deaths from lymphatic leukaemia occurred since 1970, in subjects 60 years of age and older, and with at least 30 YSFE (SMR=308, 95% CI: 84-789). Three men had 35 or more years of fire department service, while the other had worked 30-34 years (SMR for 30+ years of employment=364, 95% CI: 99-932). Records were available from the OCTRF for three of the four deaths, and all were incident cases of lymphatic leukaemia; however, only one was histologically confirmed.

Major cardiovascular diseases (SMR=99, 95% CI: 89-110)

The group of causes labelled "major cardiovascular diseases" was responsible for 381 deaths with an overall SMR of 99. The SMR hovers around 100 across all calendar periods and for all categories of YSFE and age at risk. According to duration, the SMRs are 97, 113, and 91 (Table 8), exhibiting no obvious pattern. In the analysis of five year duration categories, there is one statistically significant excess at 20-24 years worked (47 deaths, SMR=145, 95% CI: 107-193). While risk is low among current workers (83 deaths, SMR=62, 95% CI: 50-77), it is high among terminated workers (290 deaths, SMR=117, 95% CI: 104-131). Within this overall cause of death category, ischaemic heart disease accounted for 289 deaths. This gave an SMR of 104 (95% CI: 92-117), which varied little according to calendar period and YSFE. One statistically significant excess for ischaemic heart

disease occurred at 20-29 YSFE during 1975-1979, with 21 deaths and an SMR of 208 (95% CI: 129-318).

Myocardial infarction (SMR=107, 95% CI: 93-123)

Death from myocardial infarction (MI) was certified for 205 fire fighters. One statistically significant excess for this cause is apparent during 1955-1959 (30 deaths, SMR=174, 95% CI: 117-248), in part due to an excess at 40-49 YSFE within this calendar period (11 deaths, SMR=207, 95% CI: 103-371). Another statistically significant excess is apparent at 20-29 YSFE during the period 1975-1979 (15 deaths, SMR=196, 95% CI: 110-323). The SMRs do not vary substantially from 100 over the range of YSFE, while analysis by age indicates that the risk is slightly higher for those under 60 years (SMR=117, 95% CI: 94-143). With 201 subjects in the analysis by duration, there is no apparent trend, with SMRs of 108, 120 and 98 (Table 8). Within five year group of duration, however, a statistically significant excess is apparent at 20-24 years (30 deaths, SMR=165, 95% CI: 111-236). Again, risk is low among current workers (54 deaths, SMR=71, 95% CI: 54-93), and high among terminated workers (147 deaths, SMR=129, 95% CI: 109-152).

Arteriosclerosis (SMR=141, 95% CI: 91-210)

The non-statistically significant excess seen for arteriosclerosis does not exhibit any pattern according to YSFE for the 24 deaths certified to this cause. However, in two calendar periods the excess is significant: during 1960-1964 (5 deaths, SMR=347, 95% CI: 113-810) due in part to an excess at 40-49 YSFE (4 deaths, SMR=548, 95% CI: 149-1403), and during 1980-1984 (7 deaths, SMR=252, 95% CI: 101-519). Risk is slightly elevated in younger men (4 deaths, SMR=113, 95% CI: 31-289), but highest in older men with an SMR of 149 (20 deaths, 95% CI: 91-230), although confidence intervals overlap. Risk is relatively high and constant across all duration categories (Table 8): 145, 128, and 148 for less than 15, 15-29, and 30 or more years, respectively.

Diseases of the veins, lymphatics and other circulatory system (SMR=167, 95% CI: 72-330)

While diseases of the veins, lymphatics and other circulatory system (8 deaths) show a moderate excess overall, there is a statistically significant excess during the period 1965-1969 (4 deaths, SMR=769, 95% CI: 210-1970). This is due mainly to 3 deaths which occurred at 40-49 YSFE within this calendar period (SMR=1429, 95% CI: 295-4175). The SMR for the total calendar period 1950-1989 for the category 40-49 YSFE is 374 (4 deaths, 95% CI: 102-957). No death occurred before 10 YSFE or after 50 YSFE, or in those

with less than 15 years employment. Risk is elevated only for those with 15-29 years duration of employment, but is identical for current and terminated workers (SMR=169).

Chronic bronchitis, emphysema and asthma (SMR=130, 95% CI: 87-188)

While the 28 deaths certified to chronic bronchitis, emphysema and asthma do not represent a statistically significant excess overall, there is somewhat of a trend apparent according to increasing YSFE. No death occurred before 20 YSFE and the SMR for 20-29 YSFE is 80. In the next four successive 10 year periods of YSFE, the SMRs consistently increase, at 124, 137, 161 and finally 250 (not shown). This pattern is most pronounced in the calendar period 1985-1989, with SMRs rising from 0 to 150 to 179 to 291 to 1111 in the YSFE intervals. SMRs are also increased in other calendar periods. While there is low risk at less than 60 years of age (2 deaths, SMR=43, 95% CI: 5-155), the SMR for those 60 years and older is 155 (95% CI: 101-227). SMRs vary substantially over the range of employment duration (Table 8). The only death attributed to asthma was that of a man who had worked 35 years or more.

The deaths in this group of causes are the same as the ones in "chronic obstructive pulmonary disease" (COPD) except for one additional death in COPD from "other diseases of the respiratory tract." For COPD, the same pattern of consistently increasing SMRs with increasing YSFE is seen, and the overall SMR for 29 deaths is 120 (95% CI: 80-172).

Cirrhosis of the liver (SMR=133, 95% CI: 87-195)

Although cirrhosis of the liver exhibits no excess overall, there is a statistically significant SMR during 1980-1984 (11 deaths, SMR=269, 95% CI: 134-481). Of the 26 cirrhosis deaths, no death occurred before 10 YSFE, and risk is highest after 20 YSFE. The SMRs are similar for the two broad age categories (Table 7), but there is a statistically significant excess at ages 40-49 (11 deaths, SMR=212, 95% CI: 106-379). Excess risk is seen only for those working for 15-29 years (Table 8, SMR=189, 95% CI: 110-303, 17 deaths), partly due to an excess at 20-24 years (8 deaths, SMR=258, 95% CI: 111-509). There is elevated risk only among terminated workers (17 deaths, SMR=207, 95% CI: 121-332).

Gallbladder diseases (SMR=296, 95% CI: 96-690)

Gallbladder diseases account for only 5 deaths, but the overall SMR is elevated at almost 300. There is one statistically significant excess for the calendar period

1955-1959 (2 deaths, SMR=1333, 95% CI: 161-4816). All five deaths occurred at 30+ YSFE (SMR=385, 95% CI: 125-898), at 60 years or older (SMR=420, 95% CI: 136-980), and at 30+ years of employment (SMR=472, 95% CI: 153-1101).

4. DISCUSSION

Summary

This epidemiologic study suggests that fire fighting is a hazardous occupation in terms of increasing risk of death due to several causes, such as cancer of the brain and other tumours of the nervous system, "other" malignant neoplasms, and aortic aneurysm. There are also suggestive increases in risk for several other causes of death, including various cancers, and diseases of the respiratory, circulatory and digestive system.

Study limitations

As in any epidemiologic study, the associations reported here may be due to chance, bias, or confounding. Multiple comparisons were made, and factors other than fire fighting, such as smoking, alcohol or diet were not taken into account. In the absence of an apparent risk of lung cancer in these fire fighters, however, excess smoking among fire fighters is most likely not a plausible explanation of the increased risks seen for some cancers and cardiovascular diseases.

Causal relationships are rarely established in one study; in addition to the strength of an association, it is biological plausibility and consistency across several studies that will establish a causal link between an occupation and a disease. It is also important to note that an epidemiologic study cannot definitively establish the absence of a risk but can often place an upper bound on overall risk.

This study shares a common feature of mortality cohort studies of fire fighters in that no information is available on an individual's exposure. In the absence of information on individual fire fighter activity and fire hall assignment and in light of the small variation in job assignment of fire department employees, the only feasible index of exposure for this cohort study is number of years of employment. (This is discussed extensively in Appendix D.) In this respect, the results are directly comparable to those from other studies. For duration of employment to be a valid approximation to the cumulative exposure to substances experienced by fire fighters, actual exposure would have to be close to uniform over a fire fighter's

career, over calendar time and across fire fighters. Any departure from this uniformity will be ignored in the analysis by the use of years of employment as the sole index of exposure. It is known, however, that very different exposures can occur for two fire fighters at the same fire even if they are side by side (Brandt-Rauf et al., 1988). As a result, variability in risk associated with variability in exposure will go undetected in this study. Risks associated with fire fighting may be underestimated due to the misclassification which undoubtedly occurs when duration of employment is used as a proxy for exposure.

When this study was designed, there was a concern that the bias known as the healthy worker effect would mask some important risks. This bias can occur when the mortality experience of a working population is compared to that of the population at large, which comprises both those who work and those who do not or cannot work. The working population, being healthier, has a lower mortality rate for many causes than the reference population, so there is sometimes a problem in determining what exactly constitutes an excess risk for the working population. In anticipation of this problem, the protocol for this study provided for a comparison of the fire fighters to another working population, the Northern Ontario Miners' Cohort. However, no strong healthy worker effect overall is evident in this study and therefore this comparison is not necessary. Furthermore, the comparison to miners would have been subject to some severe limitations, such as the inability to compare causes of death known to be in excess in miners. In the other cohort study of Canadian fire fighters (Guidotti, 1992), with the Albertan population as a reference group, a healthy worker effect was not seen. It has been reported, however, in several studies of U.S. fire fighters (Beaumont et al., 1991; Demers et al., 1992, for example). Dr. Guidotti explains that a healthy worker effect is not seen in his study because few subjects are lost to follow-up (as in this study), there is a long follow-up period (as in this study), and the use of a provincial instead of national comparison may reduce the effect since Alberta males are generally healthier than the national average.

Fire fighting is unlike most occupations in that to become a fire fighter, one must pass a medical examination, an aptitude test and tests of physical skill. The six fire departments in this study all had approximately the same entrance requirements. Although the requirements were not so stringent in the past, to be hired, one has always had be physically fit. It does not seem unreasonable to assume that at least at the start of his career, the fire fighter is much healthier than the average person in the general population. When the physical nature of fire fighting work

is considered, it becomes apparent that to continue being a fire fighter demands a level of health higher than that of the general population.

In this study, a strong healthy worker effect is evident for those under 40 years of age. In other words, fire fighters under 40 die less frequently than comparable members of the general population. However, by the time a fire fighter reaches 40, he is no longer at a reduced risk of death when compared to the general population. Furthermore, all-cause mortality shows an increasing trend in SMRs as years since first exposure (YSFE) increases. Analysis by 10 year periods indicates that a strong healthy worker effect is evident for those with less than 10 YSFE, but not evident for those workers with more than 10 YSFE. In other words, although the fire fighters experience a lower mortality rate than comparable members of the general population, this is only so for approximately the first ten years since start of employment. After this ten year period, for many causes of death their mortality experience is similar to or higher than that of the general population. Given the pattern of a rapidly diminishing healthy workers effect within about 10 years of employment as a fire fighter, it appears that the effect of fire fighting is to increase the overall mortality of this working population so that it equals that of the general population.

If this is so, there is some difficulty in interpreting SMRs, as for some causes, an SMR of 100 is an excess, relative to an SMR of about 85 for all-cause mortality that has been shown for several working populations in comparison to the general population (Meijers et al., 1989). Therefore, depending on what one judges as the most appropriate comparison group for fire fighters, the real magnitude of some of the risks seen here may be greater than the SMR indicates. The healthy worker effect is generally more pronounced for cardiovascular disease than for cancer causes of death.

Further, a survivor effect can be postulated for this cohort. That is, adequate health is required for continuing employment and unwell workers may selectively leave the fire department before 30 years of employment. If duration is the only proxy for exposure and there is a survivor effect, a true exposure-response relationship over the range of employment duration may be masked (Robins, 1987). This may explain, for example, the increases in SMRs seen until 30+ years' duration, and the decline seen after this point for some causes of death.

Potential exposures

There is very little documentation of the prevalence of actual exposures which fire fighters receive. In an effort to obtain information specific to urban Canadian fire fighters, a group of researchers was commissioned to provide exposure data on fire fighters as part of their on-going study of cancer in Metropolitan Montreal (Siemiatycki, 1991). Exposure assessment is done by a team of industrial hygienists and chemists based on a detailed personal interview. Appendix E presents the exposure assessment for 47 fire fighters, 40 of whom were from urban areas, is seen in Appendix E. In summary, several substances were identified as exposures for the majority of fire fighters (85%-98% prevalence of exposure): polycyclic aromatic hydrocarbons (PAHs), pyrolysis fumes, aliphatic aldehydes, carbon monoxide, formaldehyde, particulates from wood and soot, nitrogen oxides, sulphuric acid, chlorinated alkanes, benzopyrene, chrysotile asbestos, sulphur dioxide, monocyclic aromatic hydrocarbons, chlorine, phosgene, engine emissions, carbon tetrachloride, solvents, alkanes, hydrogen cyanide, hydrogen chloride, plastics pyrolysis products, benzene, cyanide, and lubricating oils. In experimental studies, carbon monoxide, formaldehyde and acrolein are among chemicals identified as pyrolysis products which occur even at relatively low temperatures (Edgerley, 1980).

In a recent study which measured substances in 22 fires (15 residential, 6 training, and 1 car fire) which occurred in various urban fire departments across the U.S., carbon monoxide was the most common contaminant (Jankovic et al., 1991). Distinctions were made between measurements taken in the knockdown (bringing the main body of fire under control) and overhaul (searching for and extinguishing hidden fires), and those taken inside and outside self-contained breathing apparatus. Ranked in order of decreasing occurrence in the knockdown phase, the substances found were: carbon dioxide, carbon monoxide, ethylene, methanol, methane, acetaldehyde, acrolein, benzene, hydrogen cyanide, and hydrochloric acid. Several of these chemicals were also measured, although at lower concentrations, in the overhaul phase, which is necessary mainly for larger fires. These investigators found that masks are usually not worn during the overhaul phase and that the low concentrations found which were attributed to 'inside mask" were due either to early removal or non-use of masks and not to leakage of the masks themselves. The increased use of synthetic materials since the 1950's has increased concern about fire fighters' exposure to potentially toxic chemicals.

Comparison to previous studies

In an effort to evaluate the findings of this study in relation to the total body of knowledge available, we have compared our results to all cohort studies of fire fighters published to date. Salient points are discussed below according to cause. For all cancer, and for five specific cancer sites, a recent review summarized results from six cohort studies of fire fighters (Howe and Burch, 1990). Since that publication, four additional cohort studies of fire fighters have been published which present results on the sites of cancer covered in the review. The results of all of these studies have been combined here to give an updated view of mortality among fire fighters for the following causes of death: all cancers combined; lung cancer; colon; brain cancer and other central nervous system cancers; malignant melanoma; and multiple myeloma. This section is organized to present first the cancer sites which were reviewed by Howe and Burch, followed by other selected cancer sites, and finally non-cancer sites.

All cancer

Combining ten previous studies produces an SMR of 95 (95% CI: 90-100). This is marginally lower than the SMR for all cancers in this study of 105 (95% CI: 91-120). Thus, the overall risk of all cancer does not seem to be elevated among fire fighters.

[The Seattle cohort study (Heyer et al., 1990), included in the review by Howe and Burch, is a part of an ongoing study of fire fighters in the northwest U.S. Longer follow-up of this larger cohort will soon be published (Demers et al., 1992), and these results replace Howe and Burch's figures for Seattle in the following calculations.]

Cancer of the brain and other nervous system tumours

Six previous studies which reported brain cancer mortality combine to give an SMR of 155 (95% CI: 115-205). This is somewhat lower than the result of this study, an SMR of 200 (95% CI: 109-336). When the results of the present study are combined with the previous studies' results, the overall SMR is 164 (95% CI: 126-210). In addition, two recent studies, not included in the combined results because they are not cohort studies, have reported a possible increased risk of brain cancer in fire fighters. A study of fire fighters in Hawaii produced a proportionate mortality ratio (PMR) of 378 (95% CI: 122-1171) based on 3 deaths (Grimes, 1990), and a case-control study in Massachusetts gave a standardized morbidity odds ratio (SMOR) of 86 (95% CI: 34-

215) when the state population was used as a reference, and 152 (95% CI: 39-592) when police were used as a reference (Sama et al., 1990).

The excess of brain cancer seen in the Heyer et al. (1990) study of Seattle fire fighters was limited to those dying within 15 YSFE. The authors postulate that, given this short time frame for the development of brain cancer (although they admit this interpretation is based on small numbers), fire fighters with a pre-existing condition must be affected while others are not. However, in the larger study of Seattle and Tacoma, Washington, and Portland, Oregon, excess risk is apparent both at less than 20 YSFE and greater of equal to 30 YSFE (Demers et al., 1992). This is very similar to the results in this study where excess risk is apparent both at less than 20 YSFE and at 40+ YSFE and not during the period 20-39 YSFE, although this apparent pattern in risk is also based on small numbers. Although causal factors may differ for the two YSFE periods which exhibit high risk, the excess risk does not disappear after 20 YSFE.

A comprehensive review of brain cancer and occupational risk factors found numerous studies showing increased risk of brain cancer in a wide variety of occupational groups (Thomas and Waxweiler, 1986). The lack of exposure information in most of these studies makes it difficult to determine exactly what any of them might have in common with fire fighting. However, the authors mention, among other substances, vinyl chloride, formaldehyde, acrylonitrile, polycyclic aromatic hydrocarbons, diesel and other motor fuel exhausts, trichloroethylene, asbestos and solvents as exposures that are prevalent among many of the occupational groups with increased risk of brain cancer. A recent study has demonstrated a brain cancer excess in workers exposed to polychlorinated biphenyl (Sinks et al., 1991).

Cancer of the colon

Eighty-six deaths in the combined studies yield an SMR of 109 (95% CI: 87-135), almost double the SMR of 60 for this study (95% CI: 30-108). There is therefore no evidence for increased risk of colon cancer among fire fighters.

Lung cancer

Eight studies combine to give an SMR of 93 (95% CI: 84-103), essentially the same result as found here, an SMR of 95 (95% CI: 71-123). Not included in this review is a study by Hansen (1990) comparing the mortality of male fire fighters to civil servants and salaried employees classified as such on census information. This study found a

statistically significant increase in lung cancer for fire fighters (SMR=317, 95% CI: 117-691). Hansen also reports an excess for "non-pulmonary" cancer, but provides no details of specific cause.

Malignant melanoma

PMR studies were included in the review for this cause, so the results of the review may not be as valid as those for other causes. Nevertheless, the combined SMR/PMR value is 146 (95% CI: 96-215), and including the two deaths in present study, it is 137 (95% CI: 91-198). Thus, there is a moderate excess over all studies which does not reach statistical significance.

Multiple myeloma

The combined SMR/PMR value from the review is 133 (95% CI: 87-195), whereas this study had one death that gave an SMR of 40 (95% CI: 1-215).

Other sites of cancer

Several other sites showed suggestive increases in cancer mortality risk in this study. For cancer of the pharynx, and particularly cancer of the oropharynx, statistically significant excess risk was seen at 35 or more years of employment. Elevated mortality is also seen for cancer of the rectum and rectosigmoid junction during specific calendar periods, after 10 YSFE and after 10 years of employment. For cancer of the prostate, there was a statistically significant excess at 25-29 years duration of employment. Risk for cancer of the testis increases after 10 YSFE, but only in workers with less than 10 years of employment. Lymphosarcoma and reticulum cell sarcoma risk exhibit an excess at 10-14 years of employment, while lymphatic leukaemia occurred only in those with at least 30 YSFE and 30 years of employment.

Circulatory system diseases

The mortality risk for circulatory system diseases overall in this study is not different from that of the general population. This differs from the findings of Mastromatteo (1959) who reported an excess in City of Toronto fire fighters for deaths occurring between 1921 and 1953. This overlaps with the present study by four years of follow-up, a period in which there is no excess in our study. Coronary heart disease (CHD) was also elevated in the study by Bates (1987) of fire fighters in the City of Toronto who worked six years or more. He compared mortality rates to those for males in the City only. Follow-up was from 1949 through

1984, and of 52 deaths known through pension records, 47 death certificates were obtained and examined for any circulatory system cause of death. For the remaining deaths, cause was abstracted from newspaper or coroner reports. The resulting SMR for CHD was 173 (95% CI: 107-264) based on 21 deaths. A possible explanation for these discrepant results is the different methodologies (different inclusion criteria, method of death enumeration, method of cause of death coding, and comparison populations) used by Bates and in the present study. An unpublished survey of smoking in 1980 in the City of Toronto fire department found that about one-third of fire fighters smoked. This is approximately the same as the proportion of males in the general population who smoked and therefore, smoking has been discounted as a confounding factor. Dr. Bates is continuing work on this issue and that of possible confounding by cholesterol levels and hypertension. Evidence from the U.S. shows that fire fighters smoke less than other municipal and blue-collar workers (Gerace, 1990).

Two studies have shown increasing SMRs for circulatory system diseases with increasing years of work (Vena and Fiedler, 1987; Heyer et al., 1990, and updated in Demers et al., 1992). Guidotti found no association with duration of employment for this cause (Guidotti, 1992).

A recent review discusses the possible role of occupational factors in the development of cardiovascular diseases (Kristensen 1989a and b). Some non-chemical occupational factors which could contribute to cardiovascular disease risk in fire fighters are sudden exertion during waiting or sleeping periods, environmental stress (heat, cold, noise), physical stress (from heavy equipment, prolonged work), and emotional stress (critical incidents such as witnessing fire victims, response to emergencies, responsibility for the lives of others). Substances which may potentiate a cardiovascular reaction in fire fighters include carbon monoxide and dioxide, acrolein, benzene, cyanide, hydrogen chloride, particulates, sulphur oxides, and organic solvents.

Myocardial infarction

A large number of fire fighters have been affected by the increased risk for acute myocardial infarction (MI) seen in specific calendar periods and time intervals since first employment. While the SMRs do not vary substantially from 100 over the range of YSFE, and there is no apparent trend by duration of employment, risk is highest for those under 60 years. Although the overall SMRs for acute MI are not statistically remarkable, they are higher than would be expected from a population whose work requires a degree of

physical fitness higher than that of the general population. While heavy labour may be a risk factor for MI, confounding by diet or by work related psychosocial factors is be possible (Vingard et al., 1991).

For ischaemic heart disease as a whole, Hansen (1990) reports a slight excess, while most cohort studies of fire fighters which use general population comparisons do not show any evidence of an increased mortality risk for ischaemic heart disease or myocardial infarction.

Aortic aneurysm

A statistically significant excess risk was seen in this study for death from aortic aneurysms, particularly for those with 20+ YSFE, in those 60 years of age and older, and for those who worked 25 years or more. Smoking, hypertension, diabetes, atherosclerosis, trauma and stress have been identified as possible risk factors (Lindsay and Hurst, 1979).

Chronic obstructive pulmonary disease (COPD)

This group of diseases includes any disorder, such as chronic bronchitis, chronic airway obstruction and emphysema, which is marked by persistent slowing of bronchial air flow (Higgins and Thom, 1988). There is some disagreement over whether or not asthma should be included in the definition, and in this study the one death from asthma is included. Non-occupational risk factors include smoking, air pollution, exposure to open fires (cooking), while some researchers contend that occupational factors include toluene diisocyanate, grain, wood and hardrock dust, and several irritant gases (Becklake et al., 1988). While chronic changes in pulmonary function have been documented in fire fighters (Sparrow et al., 1982; Peters et al., 1974), as has acute airway responsiveness (Sherman et al., 1989), many investigators contend that this is not reflected in cohort studies of mortality because of the healthy worker effect (Becklake et al., 1988; Rosenstock et al., 1990). In comparison to police officers, one study has shown that fire fighters experience an excess risk of non-malignant respiratory diseases (SMR=141, 95% CI: 106-184) (Rosenstock et al., 1990). In the present study, risk of death from COPD increases with increasing YSFE, indicating a diminishing healthy worker effect. Further, in the most recent calendar period, there is almost a statistically significant increase (SMR=206, 95% CI: 99-379). In the study of fire fighters in Edmonton and Calgary, COPD was moderately increased (SMR=157, 95% CI: 79-281), but showed no relation to duration of employment (Guidotti, 1992).

Cirrhosis

For cirrhosis of the liver, some elevation in mortality was seen, with highest risk after 20 YSFE and for those working for 15-29 years. Digestive system diseases as a whole were elevated in San Francisco fire fighters due to excesses of cirrhosis (SMR=227, 95% CI: 127-192) and diseases of the stomach and duodenum and the authors did not know whether to attribute this excess to fire fighting exposures, alcohol or their interaction (Beaumont, 1991).

Gallbladder diseases

Risk of death from this cause was somewhat elevated in this study, with very high risk for workers with 30+ YSFE, 30+ years of employment, and in those 60 years of age or older. Gallbladder diseases include cholelithiasis (gallstones), cholecystitis (inflammation of the gallbladder), and haematobilia (bleeding into the biliary passages). Known risk factors include obesity, trauma and a diet high in cholesterol (Schoenfield, 1977). We are not aware of any occupational risk factors that have been identified for mortality from these diseases, and results for this cause of death have not been reported in other studies of fire fighters.

5. CONCLUSION

As one of the largest investigations of fire fighters, this study has excellent power to detect and label as statistically significant even moderate increases in risk for most specific causes of death. For example, this study has demonstrated a statistically significant increase in risk of brain cancer, whereas several other studies have shown increases which have been less convincing. For at least one disease of the circulatory system, aortic aneurysm, there is an unambiguous excess. For several other causes of death we have argued that, because fire fighters are selected for their excellent health, where moderate increases are apparent compared to the general population, these are probably underestimates of the true hazards of fire fighting.

In future, a better understanding is needed of the potential exposures that fire fighters experience so that measures can be taken to reduce them. If fire fighters knew more about the exposures they were facing at each fire, preventive measures specific to those exposures could then be taken. Perhaps consideration should be given to requirements that companies and large institutions furnish fire departments

with information on the substances they have on their premises, including their locations and the amounts present. Information on exposures which fire fighters have received could then be recorded in fire department records. The widespread use of synthetic materials in homes and businesses today means that all fires have the potential of producing highly toxic gases. In choosing materials for construction, furniture and decoration, wherever possible, preference should be given to materials which produce less hazardous combustion products. In terms of prevention of excess risks, although protective breathing equipment has been required recently, there is still a need to educate fire fighters about the necessity of wearing masks and keeping them on throughout the knockdown and overhaul phases of a fire. Development of lighter and less restrictive protective gear is required to provide fire fighters with as safe a working environment as possible.

REFERENCES

Bates, J.T. Coronary artery disease in the Toronto fire department. Journal of occupational medicine. Vol.29,no.2 (Feb., 1987). p.132-135.

Beaumont, J.J.; Chu, G.S.T.; Jones, J.R.; Schenker, M.B.; Singleton, J.A.; Piantanida, L.G.; Reiterman, M. An epidemiologic study of cancer and other causes of mortality in San Francisco firefighters. American journal of industrial medicine. Vol. 19 (1991) . p 357-372.

Becklake, M.R.; Bourbeau, J.; Menzies, R.; Ernst, P. The relationship between acute and chronic airway responses to occupational exposures. Current pulmonology. Vol.9 (1988). p.25-66.

Brandt-Rauf, P.W.; Fallon, L.F.; Tarantini, T.; Idema, C.; Andrews, L. Health hazards of fire fighters: exposure assessment. British journal of industrial medicine. Vol. 45 (1988). p.606-612.

Breslow, N.E.; Day, N.E. Statistical Analysis in Cancer Research, V. II: The Design and Analysis of Cohort Studies. Lyon: IARC Scientific Publications No. 82, 1987.

Chu, D. The mortality and morbidity of rare cancers, 1962-1986. Chronic diseases in Canada. Vol.12,no.3(1991). p.35-38.

Coleman, M.P.; Hermon C.; Douglas A. Person-years (PYRS): A Fortran Program for Cohort Analysis. Lyon: IARC Internal Report 89/006, 1989.

Demers, P.A.; Heyer, N.J.; Rosenstock L. Mortality among firefighters from three northwestern United States cities. British journal of industrial medicine. Vol.49 (1992) (in press).

Dibbs, E.; Thomas, H.A.; et al. Fire fighting and coronary heart disease. Circulation. Vol.65,no.5(1982). p.943-946

Edgerley, J. Oxidative pyrolysis of plastics. Fire and materials. Vol.4,no.2(1980). p.77-82.

Eliopulos, E.; Armstrong, B.K.; Spickett, J.T.; Heyworth F. Mortality of fire fighters in western Australia. British journal of industrial medicine. Vol.41,no.2(May, 1984). p.183-187.

Feuer, E.; Rosenman, K. Mortality in police and fire fighters in New Jersey. American journal of industrial medicine. Vol.9,no.6 (1986). p.517-527.

Gallagher, R.P.; Threlfall, W.J.; et al. Occupational mortality in British Columbia 1950-1978. Cancer Control Agency of British Columbia. Statistics Canada. Health and Welfare Canada. (Apr., 1986).

Gerace, T.A. Road to a smoke-free fire service for Florida: Policies and progress. Journal of public health policy. Vol.11 (1990). p.206-217.

Grimes, G.; Hirsch, D.; Borgeson, D. Risk of death among Honolulu fire fighters. Hawaii medical journal. Vol. 50, no. 3 (1991). p.82-85.

Guidotti, T.L. Mortality of urban firefighters in Alberta, 1927-1987. American journal of industrial medicine (1992) (in press).

Hansen, E.S. A cohort study on the mortality of firefighters. British journal of industrial medicine. Vol. 47. (1990). p.805-809.

Heyer, N.; Weiss N.S.; Demers, P.; Rosenstock L. Cohort mortality of Seattle fire fighters: 1945-1983. American journal of industrial medicine. Vol.17 (1990). p.493-504.

Higgins, M.; Thom, T. Epidemiology and natural history of COPD. Current pulmonology. Vol.9 (1988). p.1-24.

Howe, G.R.; Burch, J.D. Fire fighters and risk of cancer: an assessment and overview of the epidemiologic evidence. American journal of epidemiology. Vol. 132, no. 6 (1990). p.1039-1050.

Howe, G.R.; Chiarelli, A.M.; Lindsay, J. Components and modifiers of the healthy worker effect: Evidence from three occupational cohorts and implications for industrial compensation. American journal of epidemiology. Vol. 128, no. 6 (1988). p.1364-1375.

Jankovic, J.; Jones, W.; Burkhart, J.; Noonan, G. Environmental study of firefighters. Annals of occupational hygiene. Vol.35 (1991). p.581-602.

Kristensen, T.S. Cardiovascular diseases and the work environment. A critical review of the epidemiologic literature on chemical factors. Scandinavian journal of work, environment and health. Vol. 15 (1989a). p.245-264.

Kristensen, T.S. Cardiovascular diseases and the work environment. A critical review of the epidemiologic literature on nonchemical factors. Scandinavian journal of work, environment and health. Vol. 15 (1989b). p.245-264.

Lindsay, J.; Hurst, J.W. The Aorta. New York: Grune and Stratton,

Inc., (1979).

Mastromatteo, E. Mortality in city firemen-- II. A study of mortality in firemen of a city fire department. A.M.A. archives of industrial health. Vol.20 (Sept., 1959). p.227-233.

Meijers, J.M.M.; Swaen, G.M.H.; Volovics, A.; Lucas, L.J.; Van Vliet, K. Occupational cohort studies: The influence of design characteristics on the healthy worker effect. International journal of epidemiology. Vol.18,no.4 (1989). p.970-975.

Milham, S. Occupational mortality in Washington State 1950-1979. U.S. Department of Health and Human Services. Public health service. Center for disease control. National institute for occupational safety and health. Cincinnati, Ohio. (Oct., 1983).

Morton, W.; Marjanovic, D. Leukaemia incidence in the Portland-Vancouver metropolitan area. American journal of industrial medicine. Vol.6,no.3 (May, 1984). p.185-205.

Musk, A.W.; Monson, R.R.; Peters, J.M.; Peters, R.K. Mortality among Boston fire fighters, 1915-1975. British journal of industrial medicine. Vol.35,no.2 (May, 1978). p.104-108.

Peters, J.; Theriault, G.; Fine, L.; Wegman, D. Chronic effects of fire fighting on pulmonary function. New england journal of medicine. Vol.291 (1974). p.1320-1322.

Raman, S. Mortality analysis using Monson's Computer Program. Draft Report, Statistics Canada (March, 1985).

Robins, J.M. A graphical approach to the identification and estimation of causal parameters in mortality studies with sustained exposure periods. Journal of chronic diseases. Vol.40(Suppl.2) (1987). p.139S-161S.

Rosenstock, L.; Demers, P.; Heyer, N.J.; Barnhart, S. Respiratory mortality among fire fighters. British journal of industrial medicine. Vol.47 (1990). p.462-465.

Sama, S.R.; Martin, T.R.; Davis, L.K.; Kriebel, D. Cancer incidence among Massachusetts firefighters, 1982-1986. American journal of industrial medicine. Vol. 18 (1990). p.47-54.

Shannon, H.; Jamieson, E.; Walsh, C.; Julian, J.; Fair, M.; Buffet, A. Comparison of individual follow-up and computerized record linkage using the Canadian National Mortality Data Base. Canadian journal of public health. Vol.80 (1989). p.54-57.

Sherman, C.B.; Barnhart, S.; Miller, M.F.; Segal, M.R.; Aitken, M.; Schoene, R.; Daniell, W.; Rosenstock, L. Firefighting acutely

increases airway responsiveness. American review of respiratory disease. Vol.140 (1989). p.185-190.

Shoenfield, L.J. Diseases of the gallbladder and biliary system. New York: John Wiley and Sons (1977).

Sidor, R.; Peters, J.M. Prevalence rates of chronic non-specific respiratory disease in fire fighters. American review of respiratory disease. Vol.109,no.1 (Jan., 1974). p.255-261.

Siemiatycki J. Risk factors for cancer in the workplace. Boca Raton: CRC Press (1991).

Sinks, T.; Smith, A.B.; Rinsky, R.; Watkins, K.; Shults, R.; Steele, G. Health Hazard Evaluation Report HETA-89-116-2094 Westinghouse Electric Corporation, Bloomington, Indiana. Cincinnati, Ohio: NIOSH (Jan., 1991).

Smith, M.E.; Newcombe, H. Use of the Canadian Mortality Data Base for epidemiological follow-up. Canadian journal of public health. Vol.73 (1982). p.39-46.

Sparrow, D.; Bosse, R.; Rosner, B.; Weils, S. The effect of occupational exposures on pulmonary function-- a longitudinal evaluation of fire fighters and non-firefighters. American review of respiratory diseases. Vol.123 (1982). p.319-322.

Thomas, T.L.; Waxweiler, R.J. Brain tumours and occupational risk factors: A review. Scandinavian journal of work, environment, and health. Vol.12 (1986). p.1-15.

Tomasian, A.; Fogarty, K.; et al. Prevalence of lung disease in Cambridge, Massachusetts, USA fire fighters. American review of respiratory diseases. Vol.125 (part 2)(1982). p.164.

Tornling, G.; Gustavsson, A.; Gustavsson, P.; Hogstedt, C.; Soderholm, M. Dodsorsaker bland brandman i stockholm. Omradesvisa undersokningar av kemiska miljoer (25: Dec., 1990).

Vena, J.E.; Fiedler, R.C. Mortality of a municipal-worker cohort; IV. fire fighters. American journal of industrial medicine. Vol.11,no.6 (1987). p.671-684.

Vingard, E.; Koster, M.; Alredsson, L.; Fellenius, E.; Hogstedt, C. Physical work load and myocardial infarction among men. Eighth international symposium: epidemiology in occupational health. Paris (Sept, 1990) (abstract). p.121.

Young, I.; Jackson, J.; et al. Chronic respiratory disease and respiratory function in a group of fire fighters. Medical journal of Australia. (June, 1980). p.654-658.

LIST OF TABLES:

1 EXCLUSIONS AND NUMBER OF FIRE FIGHTERS REMOVED DUE TO EACH EXCLUSION

2 DESCRIPTIVE STATISTICS FOR ENTIRE COHORT

3 PERSON-YEARS CLASSIFIED ACCORDING TO YEARS SINCE FIRST EXPOSURE AND CALENDAR TIME.

4 PERSON-YEARS CLASSIFIED ACCORDING TO YEARS WORKED

5 MORTALITY BY DETAILED CAUSE FOR TOTAL COHORT

6 STANDARDIZED MORTALITY RATIOS BY YEARS SINCE FIRST EXPOSURE FOR SELECTED CAUSES

7 STANDARDIZED MORTALITY RATIOS BY AGE FOR SELECTED CAUSES

8 STANDARDIZED MORTALITY RATIOS BY DURATION OF EMPLOYMENT FOR SELECTED CAUSES

9 SUMMARY OF PREVIOUS STUDIES COMBINED WITH THE CURRENT STUDY

10 REVIEW OF PREVIOUS STUDIES: ALL CANCERS

11 REVIEW OF PREVIOUS STUDIES: BRAIN AND OTHER CENTRAL NERVOUS SYSTEM CANCERS

12 REVIEW OF PREVIOUS STUDIES: LUNG CANCER

13 REVIEW OF PREVIOUS STUDIES: COLON CANCER

14 REVIEW OF PREVIOUS STUDIES: MALIGNANT MELANOMA

15 REVIEW OF PREVIOUS STUDIES: MULTIPLE MYELOMA

TABLE 1: EXCLUSIONS AND NUMBER OF FIREFIGHTERS REMOVED DUE TO EACH EXCLUSION

SIZE OF COHORT BEFORE EXCLUSION	REASON FOR EXCLUSION	NUMBER EXCLUDED
5995	DUPLICATE ENTRIES	28
5967	FEMALES	146
5821	KNOWN DEAD BEFORE 1 JAN 1950	65
5756	NOT KNOWN ALIVE AFTER 1 JAN 1950	58
5698	UNKNOWN BIRTH DATES	48
5650	UNKNOWN HIRE	24
5626	LESS THAN 6 MONTHS' EMPLOYMENT	211
5415	AGED 85 BEFORE FOLLOWUP BEGAN	1
5414	UNKNOWN TERMINATION DATES	41
FINAL SIZE OF COHORT FOR PRINCIPAL ANALYSIS		5414
SIZE OF COHORT FOR ANALYSES USING DURATION OF EMPLOYMENT		5373

Table 2: Descriptive Statistics

Variable	N	Minimum	Maximum	Mean	Std Dev
FOLLOWUP	5414	0.38	40.00	21.46	11.79
YSFE	5414	0.57	67.71	24.38	14.59
AGEHIRE	5414	16.40	61.52	25.28	3.79
AGEDEATH	837	22.77	95.83	63.29	14.96
DUR	5373	0.50	54.75	19.89	11.99

Table 3: PERSON YEARS CLASSIFIED ACCORDING TO YEARS SINCE FIRST EXPOSURE AND CALENDAR TIME

	1950-1954	1955-1959	1960-1964	1965-1969	1970-1974	1975-1979	1980-1984	1985-1989	1950-1989
0- 9	2855.74	4452.56	6509.06	6887.88	5850.17	4724.18	4740.37	5126.21	41146.18
10- 19	760.37	1854.39	2785.72	4374.39	6413.78	6797.96	5794.58	4678.16	33459.35
20- 29	656.06	632.04	723.81	1760.32	2630.24	4157.07	6154.55	6561.32	23275.41
30- 39	1140.82	1086.17	573.05	567.79	682.65	1620.06	2315.87	3791.74	11778.14
40- 49	210.00	427.86	844.13	735.52	378.95	416.09	508.95	1302.97	4824.47
50- 59	19.24	42.75	113.60	196.55	445.57	343.93	189.85	221.22	1572.70
60- 79	3.59	.49	5.69	4.77	15.38	27.32	74.68	12.79	144.72
0- 79	5645.81	8496.27	11555.07	14527.22	16416.73	18086.61	19778.86	21694.40	116201.00

Table 4:

PERSON-YEARS CLASSIFIED ACCORDING TO YEARS OF EMPLOYMENT

YEARS WORKED	NO. IN GROUP	OBSERVED	PERSON-YEARS
0- 1	4411	4	5157.40
1- 5	4586	30	19423.17
5-10	4081	48	21981.38
10-15	3470	48	18383.03
15-20	2919	54	14190.90
20-25	2476	88	12200.61
25-30	1911	98	9911.64
30-35	1292	141	7870.13
35+	514	242	6173.62
TOTAL	5373	753	115291.90

Table 5

Mortality by detailed cause for total cohort

Cause of death (ICD9)	OBS	EXP	SMR	95% CI
ALL CAUSES (000-999)	777	823.50	94	(88-101)
INFECTIVE, PARASITIC DISEASES (1-39)	1	7.21	14	(0- 77)
ALL MALIGNANT NEOPLASMS (140-208)	199	190.39	105	(90-120)
Pharynx (146-149)	4	2.87	139	(38-357)
Oesophagus (150)	2	5.04	40	(5-143)
Stomach (151)	7	13.79	51	(20-105)
Colon (153)	11	18.25	60	(30-108)
Rectum and rectosigmoid junction (154)	13	7.60	171	(91-293)
Liver and bile ducts (155)	2	2.37	84	(10-305)
Pancreas (157)	14	9.98	140	(77-235)
Larynx (161)	1	2.70	37	(1-206)
Trachea, bronchus & lung (162)	54	57.08	95	(71-123)
Malignant melanoma (172)	2	2.76	72	(9-262)
Prostate (185)	16	12.09	132	(76-215)
Testis (186)	3	1.22	246	(51-719)
Bladder (188)	7	5.49	128	(51-263)
Kidney and ureter (189)	2	4.63	43	(5-156)
Brain and other nervous system (191-192)	14	7.00	200	(109-336)
Lymphatic & hematopoietic tissue (200-208)	18	18.39	98	(58-155)
Lymphosarcoma (200)	3	1.48	203	(42-592)
Hodgkin's disease (201)	1	2.19	46	(1-254)
Multiple myeloma (203)	1	2.59	39	(1-215)
Lymphatic leukaemia (204)	4	2.11	190	(52-485)
Myeloid leukaemia (205)	4	3.35	119	(33-306)
Other	5	6.67	75	(24-175)
Other malignant (195-199)	20	7.08	282	(172-436)
ENDOCRINE, NUTRITIONAL DISEASES (240-279)	5	14.61	34	(11-80)
CIRCULATORY SYSTEM (390-459)	384	387.79	99	(89-109)
Ischaemic heart disease (410-414,429.2)	289	278.38	104	(92-117)
Acute myocardial infarction (410)	205	191.65	107	(93-122)
Cerebrovascular disease (430-438)	41	54.02	76	(55-103)
Arteriosclerosis (440-448)	24	16.98	141	(91-210)
Aortic aneurysm (441)	19	8.40	226	(136-353)
Diseases of eins, etc. (415,451-459)	8	4.78	167	(72-330)
RESPIRATORY SYSTEM (460-519)	45	46.12	98	(71-131)
Chronic bronchitis, emphysema, asthma (490-493,496)	28	21.47	130	(87-189)
DIGESTIVE SYSTEM (520-579)	45	36.85	122	(89-163)
Gallbladder diseases ( )	5	1.69	296	(96-690)
GENITOURINARY SYSTEM (580-629)	11	10.41	106	(53-189)
SYMPTOMS/ILL-DEFINED (780-799)	1	6.06	17	(0- 92)
EXTERNAL CAUSES (E800-999)	70	100.72	69	(54- 88)
Motor vehicle traffic accidents (810-825)	22	31.10	71	(44-107)
Accidental falls (880-888)	9	7.82	115	(53-219)
Accidents caused by fire & flames(890-899)	4	3.23	124	(34-317)
Suicide (950-959)	18	26.68	67	(40-107)
????OTHER KNOWN CAUSES	16	22.81	70	( )

Table 6: Standardized Mortality Ratios by Years Since First Employment for Selected Causes

Cause	Years Since First Exposure								
	< 20 Years			20-29 Years			≥ 30 Years		
	Obs.	SMR	95% CI	Obs.	SMR	95% CI	Obs	SMR	95% CI
All Causes	120	79	65- 94	143	95	80-112	514	99	91-108
All Cancers	28	120	77-170	36	94	66-131	135	106	89-125
pharynx	0	0	*******	1	122	3-679	3	188	39-549
rectum	1	125	3-696	2	146	18-527	10	179	86-329
pancreas	1	100	3-557	2	95	12-344	11	157	78-281
lung	1	23	1-128	13	103	55-176	40	100	71-136
melanoma	1	91	2-507	1	130	3-724	0	0	*******
prostate	0	0	*******	2	244	30-881	14	126	69-211
testis	3	333	69-973	0	0	*******	0	0	*******
brain & other nervous system	6	286	105-623	2	99	12-356	6	222	81-483
lymphatic leukaemia	0	0	*******	0	0	*******	4	267	73-684
other malignant neoplasms	1	143	4-797	4	288	78-737	15	306	171-505
Circulatory System	37	93	65-128	77	118	93-147	267	96	85-108
acute myocardial infarction	22	102	64-154	48	126	93-167	135	102	86-121
arteriosclerosis	1	100	3-557	3	165	34-482	20	141	86-218
aortic aneurysm	0	0	*******	3	303	62-886	16	232	133-377
diseases of veins, lymphatics, etc.	1	143	4-797	2	211	25-760	5	161	52-376
Chronic bronchitis, asthma & emphysema	0	0	*******	2	80	10-290	26	146	95-214
Cirrhosis of the liver	4	87	24-223	10	152	73-280	12	141	73-246
Gallbladder diseases	0	0	*******	0	0	*******	5	385	125-898

Table 7: Standardized Mortality Ratios by Age for Selected Causes

Cause	Age					
	< 60 Years			≥ 60 Years		
	Obs.	SMR	95% CI	Obs	SMR	95% CI
All Causes	337	91	81-101	440	97	88-107
All Cancers	87	107	86-132	112	104	86-125
pharynx	1	61	2-340	3	240	49-701
rectum	4	138	38-353	9	191	87-363
pancreas	4	97	26-248	10	170	81-313
lung	22	91	57-138	32	97	66-137
melanoma	2	93	11-336	0	0	******
prostate	2	153	19-553	14	130	71-218
testis	3	268	55-783	0	0	******
brain & other nervous system	10	198	95-364			
lymphatic leukaemia	0	0	******	4	204	56-522
other malignant	9	304	139-577	4	336	92-860
Circulatory System	150	110	93-129	11	266	133-476
acute myocardial infarction	93	117	94-146	231	93	81-106
arteriosclerosis	4	113	31-289	112	100	82-120
aortic aneurysm	3	163	33-473	20	149	91-230
veins, lymphatics & other	3	148	31-433	16	245	140-398
Respiratory System	7	57	23-117	5	181	59-422
chronic bronchitis, asthma & emphysema	2	43	5-155	38	112	79-154
Digestive System	20	96	58-148	26	155	101-227
cirrhosis of the liver	18	130	77-205	24	156	100-232
gallbladder diseases	0	0	******	8	138	60-272
				5	420	136-980

Table 8: Standardized Mortality Ratios by Duration of Employment for Selected Causes

Cause	Duration of Employment								
	< 15 years			15-29 Years			≥ 30 Years		
	Obs.	SMR	95% CI	Obs.	SMR	95% CI	Obs	SMR	95% CI
All Causes	130	88	74-104	240	97	85-110	383	91	82-101
All Cancers	36	137	96-190	53	87	65-114	100	100	81-122
pharynx	1	227	6-1265	0	0	*****	3	236	49-699
rectum	0	0	*****	5	220	71-513	8	180	78-355
pancreas	2	172	21-621	3	92	19-269	9	165	75-313
lung	8	129	56-254	16	83	47-135	27	87	57-127
melanoma	1	109	3-607	1	89	2-496	0	0	******
prostate	1	152	4-847	5	213	69-497	9	101	46-192
testis	3	353	73-1032	0	0	******	0	0	******
brain & other nervous system	5	258	84-602	3	105	22-307	5	233	76-544
lymphatic leukaemia	0	0	******	0	0	******	4	364	99-932
other malignant	4	426	116-1091	3	134	28-392	13	340	181-581
Circulatory System	40	97	69-132	126	113	94-135	207	91	79-104
acute myocardial infarction	23	108	68-162	73	120	94-151	105	98	80-119
arteriosclerosis	2	145	18-254	5	128	42-299	17	148	86-237
aortic aneurysm	1	143	4-797	5	242	79-565	13	239	127-409
Respiratory System	4	80	22-205	7	62	25-128	32	109	75-154
chronic bronchitis, asthma & emphysema	3	175	36-511	4	76	21-195	19	133	80-208
Digestive System	4	59	16-151	19	135	82-256	21	134	83-205
cirrhosis of the liver	4	100	27-256	17	189	110-303	5	78	25-182
gallbladder diseases	0	0	******	0	0	*******	5	472	153-1101

Table 9: Combined Results for Selected Cancer Causes of Death

Cause	Number Studies	Observed	Expected	SMR	95% CI	p(homogeneity)
All cancers	10	1448	1500.65	97	92-102	0.06
Lung	8	362	387.56	93	84-103	0.70
Colon	7	97	96.90	100	81-122	0.04
Brain & other central nervous system	8	63	38.52	164	126-210	0.22
Malignant melanoma	8	28	20.52	136	90-197	0.34
Multiple myeloma	7	27	22.16	122	80-178	0.01

Table 10: All Cancer

Studies of fire fighters and observed and expected numbers of deaths from all cancers, combining Howe and Burch results (1990) with four recent papers and this one.

Study	No. of fire-fighters	Obs.	Exp.	All Cancer Deaths SMR	95% C.I.
Howe and Burch:					
Buffalo	1867	102	94.0	109	88-132
Boston	5655	367	427.3	86	77- 95
Toronto[1]	1832	26	27	96	63-141
Canada	1006	12	16.2	74	38-129
Western Australia	990	30	27.5	109	74-156
Los Angeles	6722	n/a	n/a	n/a	n/a
IDSP					
NW U.S	4546	291	305.84	95	85-107
San Francisco	3306	236	248.42	95	84-108
Alberta	3328	92	72.7	127	102-156
Toronto[2]	5414	199	190.39	105	91-120
Stockholm	1116	93	91.3	102	82-125
Total		1448	1500.65	96.5	92-102

$X^2(9) = 16.1486$ p(homogeneity)=0.06

1: Mastromatteo, 1959
2: The current study

Table 11: Lung Cancer

Studies of fire fighters and observed and expected numbers of deaths from lung cancer, combining Howe and Burch results (1990) with four recent sets of results and this one.

Study	Obs.	Exp.	SMR	95% C.I.
Howe and Burch:				
Buffalo	28	29.8	94	62-136
Boston	70	79.6	88	69-111
Canada	6	5.4	111	41-242
Western Australia	7	8.3	84	34-174
IDSP				
NW U.S.	95	98.95	96	77-117
San Francisco	60	71.43	84	64-108
Alberta	24	16.9	142	91-211
Toronto[1]	54	57.08	95	71-123
Stockholm	18	20.1	90	53-142
Total	362	387.56	93	84-103

$X^2(8) = 5.5738$ p(homogeneity)=0.70

1: The current study

Table 12: Colon Cancer

Observed and expected numbers of deaths from colon cancer, combining Howe and Burch results (1990) with three recent sets of results and this one.

Study	Obs.	Exp.	SMR	95% C.I.
Howe and Burch:				
Buffalo	16	8.7	184	105-298
Canada	2	1.3	154	19-556
IDSP				
NW U.S.	24	28.61	85	54-126
San Francisco	24	24.24	99	63-147
Alberta	14	8.7	161	88-271
Toronto[1]	11	18.25	60	30-108
Stockholm	6	7.1	85	31-185
Total	97	96.90	100	81-122

$X^2(6) = 13.5127$ p(homogeneity)=0.04

1: The current study

Table 13: Brain and Other Central Nervous System Cancers

Observed and expected numbers of deaths from brain and other central nervous system cancers, combining Howe and Burch results (1990) with four recent sets of results.

Study	Obs.	Exp.	SMR	95% C.I.
Howe and Burch:				
Buffalo	6	2.6	235	86-512
Boston	8	7.8	103	44-202
Canada	0	0.6	0	0-625
IDSP				
NW U.S.	22	10.5	209	131-317
San Francisco	5	6.17	81	26-190
Alberta	3	2.05	147	30-429
Toronto[1]	14	7.00	200	109-336
Stockholm	5	1.8	279	91-651
Total	63	38.52	164	126-210

$X^2(7) = 9.4405$ p(homogeneity)=0.22

1: The current study

Table 14: Malignant Melanoma

Observed and expected numbers of deaths from malignant melanoma, combining Howe and Burch results (1990) with four recent sets of results and this one.

Study	Obs.	Exp.	SMR/ PMR	95% C.I.
Howe and Burch:				
Canada	0	0.2	0	0-1844
New Jersey	4	2.10	190	52- 488
Washington State	8	3	267	115- 525
California	1	1	100	3- 557

Note: Howe includes proportional mortality studies here because only Canada and the NW U.S. report melanoma deaths.

IDSP				
NW U.S.	6	6.22	98	36- 213
San Francisco	7	4.14	169	68- 348
Alberta	0	1.1	0	0- 331
Toronto[1]	2	2.76	72	9- 262
Stockholm	n/a	n/a	n/a	n/a
Total	28	20.52	136	90- 197

$\chi^2(7) = 7.9285$ p(homogeneity)=0.34

1: The current study

Table 15: Multiple Myeloma

Observed and expected numbers of deaths from multiple myeloma, combining Howe and Burch results (1990) with four recent results and this one.

Study	Obs.	Exp.	SMR/ PMR	95% C.I.
Howe and Burch:				
Canada	2	0.2	1000	121-3610
Washington State	8	6	133	58- 263
California	2	1	200	24- 722
British Columbia	2	2.5	80	10- 289
IDSP				
NW U.S.	12	8.57	140	72- 244
San Francisco	n/a	n/a	n/a	n/a
Alberta	n/a	n/a	n/a	n/a
Toronto[1]	1	2.59	39	1- 215
Stockholm	0	1.3	0	0- 287
Total	27	22.16	122	80-178
	$x^2(6)$ = 16.8732	p(homogeneity)=0.01		

1: The current study

Notes:

1) Howe includes proportional mortality studies here because only two of the cohort studies report multiple myeloma incidence.

2) In the Northwestern U.S. study, the observed and expected deaths are for the cause "neoplasms of lymphatic and haematopoietic tissues excluding lymphosarcoma, reticulosarcoma, Hodgkin's disease, leukaemia and aleukemia". The reason is that the NIOSH classification for the category in which multiple myeloma deaths fall includes other neoplasms of lymphatic and haematopoietic tissue. An investigator in the study mentioned that this category tends "to be dominated by multiple myeloma" (Heyer, personal communication).

LIST OF APPENDICES:

A PROTOCOL FOR STUDY OF METROPOLITAN FIRE FIGHTERS

B TABLE OF CONVERSIONS FROM ICD9 CODES TO LCDC CODES

C DESCRIPTIVE TABLES

D MEMO ON INDEX OF EXPOSURE FOR FIRE FIGHTERS

E OCCUPATIONAL EXPOSURES OF FIRE FIGHTERS IN METROPOLITAN MONTREAL

APPENDIX A

PROTOCOL

EPIDEMIOLOGIC STUDY OF
METROPOLITAN TORONTO FIRE FIGHTERS

PROTOCOL

Industrial Disease Standards Panel

January 17, 1990

ACKNOWLEDGEMENTS

The authors gratefully acknowledge the comments on a previous draft of this protocol made by the reviewers listed below. The reviewers' comments have been incorporated into the present version.

. James J. Beaumont	Dept. of Occupational and Environmental Medicine Univ. of California at Davis
. Shelia Hoar Zahm	Occupational Studies Branch National Cancer Institute USA
. James M. Melius	Division of Occupational Health and Environmental Epidemiology State of NY Dept. of Health
. Linda Rosenstock	Occupational Medicine Program Dept. of Medicine University of Washington

TABLE OF CONTENTS

		PAGE
1.	INTRODUCTION	A-5
2.	LITERATURE REVIEW	A-7
3.	STUDY OBJECTIVES	A-9
4.	COHORT DEFINITION AND IDENTIFICATION	A-10
5.	ABSTRACTING INFORMATION	A-11
6.	VARIABLES FOR ANALYSIS	A-11
7.	TRACING	A-15
8.	ANALYSIS	A-16
9.	REFERENT POPULATIONS	A-18
10.	STATISTICAL CONSIDERATIONS	A-19
11.	NESTED CASE-CONTROL STUDY	A-23
12.	CONFIDENTIALITY	A-24
13.	REFERENCES	A-28

EPIDEMIOLOGIC STUDY OF METROPOLITAN TORONTO FIRE FIGHTERS: PROTOCOL

1. INTRODUCTION

The acute risks to health from fire fighting range from physical injury due to falls, burns and other accidents to the potential effects of exposure to smoke and other chemicals during fire fighting activities. Not as well documented are the subacute or chronic effects of exposure to toxic substances emanating from fires and the physical and mental stress resulting from responses to emergency situations. Beyond this range of hazards for fire fighters lies the possible risk of diseases with long latency.

A submission to the Industrial Disease Standards Panel requesting the scheduling of cardiovascular disease among fire fighters in Schedule 3 of the Workers' Compensation Act led the Panel to review this issue, to recast it and place it on the Panel's agenda. The Panel has decided to review several diseases among fire fighters for any possible risk from occupational exposures.

Dr. Ernest Mastromatteo and Dr. Terence Bates have studied the mortality experience of the Toronto fire fighting force (Mastromatteo, 1959; Bates, 1987). Both mortality studies demonstrated a modest excess in cardiovascular mortality. Other studies carried out in the United States have not found increased cardiovascular risk in their study populations (Musk et al., 1977; Vena and Fiedler, 1987). However, all of these lacked information on important risk factors such as smoking.

A study of the Metropolitan Toronto area fire fighters would yield the following benefits:

1. Estimates of the risk of mortality will be developed for a broad range of diseases, including cardiovascular disease;
2. The inclusion of five metropolitan area forces in addition to the City of Toronto fire fighters will increase both the power of the study and the applicability to Ontario urban, professional fire fighters;
3. The study will examine characteristics of the fire fighter force which may affect occupational risk (duration of employment, type of work, etc.) and in this way will provide a better understanding of their relationship to the outcome being examined (mortality);
4. The Panel will use the study findings to support the development of policy for the compensation of professional fire fighters based on data acquired among its members;
5. This study will examine the largest cohort of fire fighters in a Canadian metropolitan area, thus providing a large population on which to base the development of compensation policy.

2. LITERATURE REVIEW

Acute health effects from fire fighting have been documented in both observational studies of fire fighters as well as in experimental studies in the work setting and in the laboratory. Observational studies have measured the levels of certain chemicals to which fire fighters are exposed. Experimental studies have demonstrated the physiologic concomitants of exposure to certain chemicals such as carbon monoxide (carboxyhemoglobin levels in the circulating blood), as well as those physiologic responses which are associated with the stress reaction (viz. elevated heart rate, elevated blood pressure, elevated catecholamine levels, electrocardiographic and enzyme changes).

The long-term, biologically coherent health effects from conditions of exposure in fire fighting have been studied by means of observational epidemiologic studies. The postulated effects include premature death from cardiovascular and respiratory disease and from certain cancers. The observational and experimental work documenting the stress reaction, as well as the documented exposure to certain chemicals which may cause cardiovascular injury (carbon monoxide), suggest a risk of premature death from cardiovascular disease. The hypothesis of premature death from respiratory disease and cancer is based on the detection and measurement of certain chemicals in the fumes of fires which may have respiratory toxicity and/or carcinogenic potential.

In two cross-sectional studies reporting the prevalence of chronic bronchitis (CB) and chronic obstructive airway disease (COAD) in fire fighters, no control group was used (Young, 1983; Tomasian, 1982). However, a higher prevalence of both conditions was found in fire fighters who smoked. A third study found a similar prevalence of CB among smoking fire

fighters and policemen (Sidor, 1974). Non-specific respiratory disease rates were proportional to dose as measured by various surrogates of exposure such as years employed, number of fires attended, number of times overcome by smoke, need for oxygen, etc.

A ten year follow-up study of heart disease among Boston fire fighters revealed that all age groups experienced lower rates of coronary heart disease (CHD) and myocardial infarction than a non-fire fighting comparison group followed similarly (Dibbs et al., 1982).

Proportional mortality studies using various data sources have reported findings from British Columbia (Gallagher, 1986), Washington state (Milham, 1983) and New Jersey (Feuer and Rosenman, 1984). Risk estimates based on the proportional mortality ratios (PMRs) for arteriosclerotic heart disease in police and fire fighters vary in each study. Moreover, the results are inconsistent since a slightly elevated PMR was found for employed fire fighters but not for retired ones (Feuer and Rosenman, 1984).

Morbidity studies have been inconclusive mainly because some do not provide comparison groups. One study which did use comparisons showed that fire fighters did not have higher morbidity (Dibbs, 1982). In another study, an excess of leukaemia based on four cases was found (Morton, 1984). Similar findings, however, were demonstrated for twenty other occupational categories. Several studies utilizing PMRs have not demonstrated a significant excess in mortality from cardiovascular disease, cancer or respiratory disease. However, the use of the PMR to test disease association with exposure is limited.

Of the five cohort studies published, two in Toronto

demonstrated an excess risk of cardiovascular disease (Mastromatteo, 1959; Bates, 1987); the others did not (Musk, 1978; Eliopolus, 1983; Vena and Fiedler, 1986). However, SMRs for circulatory disease mortality increased with increasing duration of employment, with an SMR of 110 in the highest duration category. Colon and bladder cancer were significantly elevated in this cohort, and elevated SMRs were seen for all causes of death and cancers of all sites combined for fire fighters with more than 40 years experience, and for fire fighters with over 45 years since first employment.

Deficits in mortality (i.e. fewer observed deaths than expected) from all causes have been demonstrated in several occupational cohort studies. This is attributed to the healthy worker effect (HWE), defined as the tendency for cohorts of employed workers to have a more favourable mortality or morbidity experience than the general population. This effect can be attributed to the fact that the general population includes people who are screened from employment due to sickness and disabilities. In order to assess the mortality experience in fire fighters, important issues such as the HWE must therefore be considered. In fact, since pre-employment medical screening is required for fire fighters, the HWE may be stronger than in some other occupational groups for whom this is not required. Two studies have attempted to account for a possible HWE by using a police force as the comparison to fire fighters (Feuer and Rosenman, 1986; L. Rosenstock, personal communication, 1989).

3. STUDY OBJECTIVES

The purpose of this study is to determine whether or not there is excess mortality among Metropolitan Toronto fire fighters. Excess mortality is defined as death from any specific cause that is significantly above expectation as determined from the cause-specific mortality rates in a reference population.

The type of study to be conducted is a historical cohort study. The mortality experience of Metropolitan Toronto fire fighters will be compared to two populations: 1) the general population of Ontario; and, 2) a cohort of Ontario miners. Like fire fighters, miners must be physically robust and therefore provide a comparison which is not affected by the HWE as the general population comparison may be.

4. COHORT DEFINITION AND IDENTIFICATION

The study will include the six fire departments within Metropolitan Toronto (cities of Toronto, North York, Scarborough, Etobicoke, York and the Borough of East York), and will cover the calendar period 1955 through 1987. The year chosen to initiate the cohort, 1955, is two years after Metropolitan Toronto was founded. Interviews with management, personnel department representatives in each fire department and responses on preliminary questionnaires indicate that personnel lists will be available to provide a complete nominal roll of workers employed in fire departments as of January 1, 1955. The cohort of fire fighters for this study will be established by creating a nominal roll of all employees of the six fire departments employed for six or more consecutive months on or after January 1, 1955 and before January 1, 1988. Six months corresponds approximately to the training period for recruits (which is 6 weeks at the Fire Academy followed by 16 weeks at a fire department). Care will be taken to insure that records are obtained from the seven previous municipalities' fire departments (Forest Hill Village, Leaside, Long Branch, Mimico, New Toronto, Swansea, Weston) which were incorporated in to Metropolitan Toronto in 1967. The cohort will be followed through December 31, 1987 for ascertainment of their mortality experience. The anticipated size of the nominal roll is approximately 4,100 fire fighters.

5. ABSTRACTING INFORMATION

A standard data collection instrument has been developed so that all information can be entered easily into a computer data base. Four data abstractors will be briefed on issues of confidentiality. Each will be required to take an oath of secrecy. They will be trained and checked for accuracy and inter-observer reliability.

The quality of fire department records will vary depending on the department, historical time period and condition of the information (i.e. cards, files, microfilm or fiche, computerized data, etc.). The records for all fire departments are available from 1955 to 1987. All departments maintain paper files (current and archives) and some current information is stored on computer. Records are located in fire department headquarters, personnel offices and archives.

6. VARIABLES

Information has been obtained from each fire department regarding the type of data available, their completeness and how they are stored. All fire departments keep information on each fire fighter's name, sex, date of birth, date of hire, date of termination, reason for termination, current and past job titles, assignments (to a particular division), social insurance number and other identifying numbers such as employee number. Only two of the fire departments keep information on the location of fire fighter's assignments. No department keeps information on fire fighters' smoking status. See Table 1 for details.

Table 1

RESULTS OF QUESTIONNAIRES SENT TO

METROPOLITAN TORONTO FIRE DEPARTMENTS

	Toronto	North York	Scarborough	Etobicoke	York	East York
name	Y	Y	Y	Y	Y	Y
sex	Y	Y	Y	Y	Y	Y
date of birth	Y	Y	Y	Y	Y	Y
date of hire	Y	Y	Y	Y	Y	Y
termination date	Y	Y	Y	Y	Y	Y
reason for term.	Y	Y	Y	Y	Y	Y
job titles	Y	Y	Y	Y	Y	Y
assignments	Y	Y	Y	Y	Y	Y
assignment location	Y	N	Y	division only	N	N
SIN	Y	Y	Y	Y	only on WCB	Y
other numbers	Y	Y	Y	Y	Y	Y
smoking status	N	N	N	N	N	N
exposures	little	little	little	little	little	little
how stored	paper	varied	varied	varied	varied	varied
completeness	good	good	good	good	good	good
where files can be found	personnel archives	fire dept. archives	fire dept. personnel archives	personnel	fire dept. archives	fire dept. personnel

In addition to the demographic variables mentioned above, several temporal variables will be available or can be calculated from the available information, including age at hire, age at exit from the cohort, calendar year of hire and exit, time since first employment, and time since last employment.

Information on many variables relating to the measurement of occupational exposure will be gathered to develop an exposure profile for each fire fighter. Variables which will serve as surrogates of exposure or components of exposure measures for individual fire fighters are duration of employment, job assignments (see below), and possibly location of assignments (there are 77 fire stations in Metropolitan Toronto). At the level of the six fire departments, it will be possible to collect information on number of alarms per year, number of unit responses per year, use of diesel apparatus, and response type (fire, vehicle fire, rubbish fire, washdown (hazardous products), spill or leak of dangerous goods, accidents without fires, etc.). Within the response type "fires" (those classified as causing dollar loss, i.e. excluding rubbish fires, for example), statistics are available on property classification such as residential, assembly, institution, commercial, manufacturing/processing, warehouse and storage, vehicles, and other. Actual fires, including those not causing dollar loss, comprise about 10%

of a department's total number of responses. As well, information on the dates of introduction of respiratory protection and the type (self-contained vs other types) is available from all fire departments. More specific measures of exposure at the individual level, such as attendance at particularly severe fires, will be gathered if the records allow. On review of fire departments' records, this may be possible only for sub-groups of the cohort (i.e. certain departments for certain years). Efforts will be made to develop composite measures of exposure (such as a ranked index of potential exposure) based on the available information recorded.

In terms of more specific job assignment information, fire department employees are in general assigned to one of six divisions: 1) administration, 2) fire prevention, 3) training, 4) repairs and maintenance, 5) communications, and 6) fire fighters (who comprise about 91% of the staff). Fire department employees tend to keep their assignments throughout their career, except for administrators who generally come from fire fighting careers. Changes in career stream are generally well documented in personnel files. Senior management (fire chief, associate fire chief) usually rise from the ranks of fire fighting.

7. TRACING

The nominal roll of Metropolitan Toronto fire fighters' names and personal identifiers will be sent to Statistics Canada (SC). Determination of vital status and cause of death for each fire fighter during the 1955 -1987 time period will be determined through record linkage with the National Mortality Data Base (NMDB) maintained by SC (Smith and Newcombe, 1982). This data base contains the death certificates of all deceased Canadian residents from 1950 to the present, as reported to SC by the Registrars of Vital Statistics for each province and the territories. A search of the NMDB results in the absolute number of deaths and underlying causes of death among members of the study population.

The NMDB computerized record linkage procedure (CRL) performs very well when provided with full name and date of birth. One study compared the performance of CRL with individual follow up using company files, driver's licence records and interviews in a cohort of 2469 men (Shannon et al., 1989). CRL was able to locate the deaths of three men untraced through individual follow-up. Contradictory vital status occurred in five men, and four these differences were resolved in favour of the CRL. Another study estimated that mortality could be underestimated by about 5%, mainly due to emigration from Canada of decedents. The CRL therefore is between 95% and

100% accurate, reliable, and complete.

Specifically for this study, a data file will be created containing information on each member of the cohort (including personal identifiers as well as complete records on all variables of interest for each fire fighter). Personal identifiers will include first name, middle initial, last name, date of birth, sex and social insurance number. With identifiers in addition to name and date of birth, the probability of correct matching in CRL is enhanced.

Once SC has completed the matching process, all personal identifiers are stripped from the data file and replaced with the vital status, and, if deceased, the ICD code and cause of death of each cohort member. The matched, and now anonymous, information will be sent back to the Panel for statistical analysis. SC will also save a file with personal identifiers which contains information necessary for the conduct of a nested case-control study. This information can be retrieved at a later time should a nested case-control study proceed.

8. ANALYSIS

The analysis will be limited to males, as small numbers of females necessitate their exclusion. Most of the Metropolitan Toronto fire departments' population is male, with only a few women hired in recent years. Age-, calendar period-, and

cause-specific mortality for Metropolitan Toronto fire fighters will be compared to that of the Ontario male population. To accomplish this, stratum-specific person years will be calculated with the Person-Years program using a modified life-table approach (Coleman et al, 1989). In calculating PYRs, no censoring at old age will take place, and the date of entry for tabulation of PYRs starts at the beginning of the observation period (January 1, 1955), or on day one of the seventh month of employment if this occurred after the start of the observation period. Observed deaths are divided by expected deaths which are computed by this program by multiplying the PYRs in each age-, and five year calendar period-specific strata by the Ontario provincial mortality rates. This ratio multiplied by 100 gives the standardized mortality ratio (SMR). Thus, an SMR greater than 100 may indicate an excess risk of death due to a particular cause in fire fighters. The statistical significance of an apparent excess will be judged through the calculation of a 95% confidence interval (CI) for each SMR, based on the assumption of an underlying Poisson distribution. When expected numbers are very small, asterisks (****) are printed to denote an upper CI which exceeds 9,999.

An analysis without exclusions on the basis of latency will be performed. As well, an investigation will take place of the effect of various latent periods, with exclusions of PYRs and

deaths based on time since first employment.

9. REFERENT POPULATIONS

The mortality of fire fighters will be compared to that in two referent populations: 1) Ontario males, and 2) Ontario miners (all males). Both comparisons will be age- and calendar period specific. The Ontario mining master file contains over 50,000 miners, divided into six main groups pn the basis of the ore type: gold, nickel-copper, iron, uranium, mixed, and other. With the present follow-up, the cohort has amassed approximately one million PYRs. For these groups combined, the SMRs are as follows: all causes SMR=100; malignant neoplasms SMR=96; circulatory system SMR=81; within circulatory, ischemic heart disease SMR=81, cerebrovascular SMR=83, and other circulatory SMR=79. This cohort therefore demonstrates deficits for circulatory system mortality compared to Ontario males, and are evidence for the HWE for these causes of death. The comparison of fire fighters to miners may therefore be free of this effect. Since uranium, nickel and gold miners have exhibited excess mortality from lung cancer, non-malignant respiratory disease, and a suggestive increase of stomach cancer, and comparisons for these causes will not be made.

Internal comparisons between sub-groups of fire fighters based on temporal and exposure variables can also be carried out to reduce the HWE. For this purpose, Poisson regression will be

used. This analysis uses observed deaths and the PYRs distribution for cross-classified categories of temporal and exposure variables and performs an internal comparison to the baseline categories (i.e. within the cohort only) (Breslow and Day, 1987).

10. STATISTICAL CONSIDERATIONS

A cohort size of approximately 4,100 fire fighters is anticipated. Assuming an average follow-up period of just over 18.5 years per fire fighter, it can be estimated that the cohort will accumulate approximately 76,400 person years (PYRs) during the study period 1955-1987.

The number of fire fighters in each of the six Metropolitan Toronto fire departments was estimated for the study start and stop years, 1955 and 1987. A pattern of growth was assumed for each department between these two years based on assumptions about residential/commercial growth and other known characteristics for each jurisdiction. The number of PYRs was calculated for each department for each study year. Mortality rates (in units of number of deaths per 100,000 population) by cause of death for Ontario males from 1955-1987 was obtained. These data were recorded by 5 year age and calendar period groups. The number of PYRs for each fire department was calculated in each calendar period, and an average fire fighter's age was derived assuming a normal aging

force with the normal number of entrants, resignations, retirees and deaths. The PYRs and average ages were combined to obtain an overall PYR and average age for the entire Metropolitan Toronto fire fighter force for each 5 year calendar period. The mortality rates covered the following causes of death: all cancers, lung cancer, non-malignant respiratory disease and cardiovascular disease. A curve relating age group to mortality rate was fit to the data for each cause of death and calendar period so that the mortality rate for any age in any given calendar period could be estimated. Substitution of the average age calculated for each 5 year period into the appropriate equation yielded an approximate mortality rate. This mortality rate was then multiplied by the corresponding number of PYRs to obtain the estimated number of expected deaths for the entire Metropolitan Toronto force.

Due to the uncertainty of the age distribution of the force, three different age scenarios are proposed, and expected numbers of deaths have been calculated for each scenario. The average ages over the study period were 44.3 years for scenario 1, 47.3 years for scenario 2 and 50.3 years for scenario 3.

The results are shown in Table 2. Each cause of death is shown for each scenario with their respective expected number of

deaths and the minimum SMR that could be detected with statistical significance. For these calculations, an alpha level of 0.05 (two-tailed), and a power of 90% was assumed.

TABLE 2

EXPECTED NUMBER OF DEATHS AND

MINIMAL DETECTABLE STANDARDIZED MORTALITY RATIO (SMR)

BY CAUSE OF DEATH AND AGE SCENARIO

Scenario	Cause of death	Exp	SMR
1	all cancers	59.12	142
	lung cancer	14.97	184
	respiratory disease	9.31	206
	cardiovascular disease	203.70	123
2	all cancers	88.80	134
	lung cancer	27.96	161
	respiratory disease	13.27	189
	cardiovascular disease	325.55	118
3	all cancers	133.49	128
	lung cancer	52.44	145
	respiratory disease	18.97	174
	cardiovascular disease	526.07	114

11. NESTED CASE-CONTROL STUDY

Whereas cohort studies identify individuals on the basis of exposure and follow individuals to an outcome (death, in this case), case-control studies identify individuals on the basis of disease (or death from a specific cause, defining the cases) and study their potential exposures as risk factors. Controls are often matched to cases on characteristics such as age and sex. The history of both cases and controls is examined in order to determine the relationship between both environmental and occupational factors and the risk of disease.

A nested case-control study is one in which both cases and controls are drawn from an existing cohort, in this case, of workers. Nested case-control studies are performed when it is necessary to collect information in addition to that already known for the cohort members. For example, in the present study, smoking status of individuals is not known from fire department records. Cases will be drawn as they occur throughout the follow-up, and controls will be identified as those "at risk" (i.e. working in the fire departments) at the time of the cases' death, matched by age, sex, and fire department. A computer program which selects controls from a cohort according to these conditions will be used (Beaumont et al., 1989).

A nested case-control study within the cohort of Metropolitan Toronto fire fighters will be performed if all of the following criteria are met:

1) The cohort analysis identifies a statistically significant excess of a specific and well-defined cause of death;

2) There are sufficient numbers of cases for investigation, judged to be at least 50 deaths;

3) There are risk factors for which information can be gathered in a reliable manner through interviews with subjects and/or next-of-kin.

For example, if the excess risk occurs for cardiovascular death, then information on risk factors such as smoking and alcohol habits would be collected from cases and controls. This information will not be available from fire department personnel files nor is it likely to appear in a consistent manner in personal physician files or in company medical files. This information can be obtained through interviews with fire fighters or their next-of-kin if they are deceased.

12. CONFIDENTIALITY

This epidemiologic study will be conducted in full compliance with the Freedom of Information and Protection of Individual Privacy Act (FOI Act). This act does not apply to municipal corporations and therefore does not deny Panel researchers access to employment records of the fire departments of Metropolitan Toronto. When the information is obtained, it

will be kept under the strictest confidence. Paper files will be kept under lock and key, and computerized information will be kept under high security, with the use of a rotating password system. The access to data will be extremely restricted. All information will be available only to Panel researchers. The researchers are bound by section 102(1) of the Worker's Compensation Act of Ontario, which states that

> "No officer of the Board and no person authorized to make an inquiry under this Part shall divulge or allow to be divulged, except in the performance of his duties or under the authority of the Board, any information obtained by him or that has come to his knowledge in making or in connection with an inspection or inquiry under this Part.".

The FOI Act restricts Panel researchers in the use they are able to make of personal information. An exception in the Act allows personal information to be disclosed to SC. This will be necessary in the course of the study, since the vital status of all fire fighters will need to be determined. Fire fighters' identifying information will be linked with the National Mortality Data Base maintained by SC. This will enable the determination of each fire fighter's vital status; and for the deceased, their date and cause of death. No further disclosure of personal information is required in the conduct of the cohort study. The file provided to SC must also contain all the information on variables which will be used in the analysis, since SC returns a file which is stripped of all personal identifiers (name, S.I.N., etc.).

This file is then used for the analysis which produces statistics with no personal information.

The disclosure of medical information maintained by physicians cannot be made without the consent of the patient because of the provisions in the Health Disciplines Act, and the general duty of a physician to maintain medical information in confidence. A consent form signed by every fire fighter would be necessary to obtain a medical report from his/her physician. No direct access to the actual physician's records is permitted except with the consent of both the fire fighter and the physician. Thus, it has been decided to drop from further consideration the issue of accessing medical records for every fire fighter. However, it is unlikely that these records would provide detailed risk factor information in a consistent and reliable manner.

If a nested case-control study is implemented, investigators need to access the names of cases and controls in order to seek an interview from them or from their next-of-kin. This release of personal information must be approved by special request from the Registrar General of the province in which the decedent expired (since the Province retains death certificates). The release of this personal information for cases and deceased controls would have to be requested from each Registrar General, which, if approved, allows SC to

release the information. Once the names are available, live subjects and next-of-kin for deceased subjects would have to be traced. The primary tracing source will be municipal pension fund information. Current address and next-of-kin information is generally available from personnel files. In addition, for live controls and Ontario residents, tracing can be done through the Ontario Ministry of Transportation and Communication (licence bureau) to obtain current addresses if these people are no longer working for the fire department or are not receiving benefits. Provisions will be made in the setting up of the data to facilitate the potential implementation of a nested case-control study.

BIBLIOGRAPHY

Bates, J.T. Coronary artery disease in the Toronto fire department. Journal of occupational medicine. Vol.29,no.2(Feb.1987). p.132-135.

Beaumont, J. An epidemiological study of mortality from cancer and other causes of death in San Francisco fire fighters. Draft report. December, 21, 1988

Beaumont, J.J.; Steenland, K.; Minton, A.; Meyer, S. A computer program for incidence density sampling of controls in case-control studies nested within occupational cohort studies. American Journal of Epidemiology (1989). vol.129 no.1 p.212-219.

Breslow, N.E.; Day, N.E. Statistical Analysis in Cancer Research, V. II: The Design and Analysis of Cohort Studies. Lyon: IARC Scientific Publications No. 82, 1987.

Coleman, M.P.; Hermon C.; Douglas A. Person-years (PYRS): A Fortran Program for Cohort Analysis. Lyon: IARC Internal Report 89/006.

Dibbs, E.; Thomas, H.A.; et al. Fire fighting and coronary heart disease. Circulation. Vol.65,no.5(1982). p.943-946

Eliopulos, E.; Armstrong, B.K.; et al. Mortality of fire fighters in western Australia. British journal of industrial medicine. Vol.41,no.2(May 1984). p.183-187.

Feuer, E.; Rosenman, K. Mortality in police and fire fighters in New Jersey. American journal of industrial medicine. Vol.9,no.6(1986). p.517-527.

Gallagher, R.P.; Threlfall, W.J.; et al. Occupational mortality in British Columbia 1950-1978. Cancer Control Agency of British Columbia. Statistics Canada. Health and Welfare Canada. (Apr.1986)

Hoar Zahm, S. Mortality Study of Philadelphia fire fighters. Protocol. February, 1987.

Mastromatteo, E. Mortality in city firemen II. A study of mortality in firemen of a city fire department. A.M.A. archives of industrial health. Vol.20(Sept.1959). p.227-233.

Milham, S. Occupational mortality in Washington State 1950-1979. U.S. Department of Health and Human Services. Public health service. Center for disease control. National institute for occupational safety and health. Cincinnati, Ohio. Oct. 1983.

Morton, W.; Marjanovic, D. Leukaemia incidence in the Portland-Vancouver metropolitan area. American journal of industrial medicine. Vol.6,no.3(May 1984). p.185-205.

Musk, A.W.; Monson, R.R.; et al. Mortality among Boston fire fighters, 1915-1975. British journal of industrial medicine. Vol.35,no.2(May 1978). p.104-108.

Raman, S. Mortality analysis using Monson's Computer Program. Draft Report, Statistics Canada, March 1985.

Rosenstock, L.; Demers, P.; et al. Northwest fire fighter study, 1945-1983. Preliminary report. September, 1987.

Shannon, H.; Jamieson, E.; Walsh, C.; Julian, J.; Fair, M.; Buffet, A. Comparison of individual follow-up and computerized record linkage using the Canadian National Mortality Data Base. Canadian Journal of Public Health. Vol.80(1989). p.54-57.

Sidor, R.; Peters, J.M. Prevalence rates of chronic non-specific respiratory disease in fire fighters. American review of respiratory disease. Vol.109,no.1(Jan. 1974). p.255-261.

Smith, M.E.; Newcombe, H. Use of the Canadian Mortality Data Base for epidemiological follow-up. Canadian Journal of Public Health Vol.73(1982). p.39-46.

Tomasian, A.; Fogarty, K.; et al. Prevalence of lung disease in Cambridge, Massachusetts, USA fire fighters. American review of respiratory diseases. Vol.125(4 part 2)(1982). p.164.

Vena, J.E.; Fiedler, R.C. Mortality of a municipal-worker cohort; IV. fire fighters. American journal of industrial medicine Vol.11,no.6(1987). p.671-684.

Young, I.; Jackson, J.; et al. Chronic respiratory disease and respiratory function in a group of fire fighters. Medical journal of Australia. (June 1980). p.654-658.

APPENDIX B

CONVERSION FROM ICD CODES TO LCDC CODES

Conversion of ICD

to LCDC Disease Codes

February, 1987

Occupational and Environmental Health Research Unit

Vital Statistics and Health Status Section

R.H. Coats Building, Station 18-A

Statistics Canada

Tunney's Pasture

Ottawa, Ontario

K1A 0T6

The Canadian Mortality Data Base file has underlying cause of death coded in machine-readable form according to the World Health Organization International Classification of Diseases (ICD). The ICD revisions used have varied over the years. Specifically, the revisions used on the Mortality Data Base are:

1950-1957	= 6th ICD
1958-1968	= 7th ICDA (American Adapted)
1969-1978	= 8th ICD
1979-present	= 9th ICD

In health studies spanning a wide range of years, it is often convenient to map the various ICD revisions to a common number for a specified disease condition so it can be studied over the total time period. The attached sheets give 174 codes that have been fequently used in studies by the Laboratory Centre for Disease Control (LCDC) at Health and Welfare.

When the Monson routine is used to analyze a cohort file at Statistics Canada, Canadian rates have been calculated for the attached 174 LCDC code conditions. The attached sheets indicate the specific ICD original codes for the various revisions which were used to map into the 174 LCDC codes.

Conversion of ICD to LCDC Diseases Codes.

Description	LCDC Code	9th ICD 1979-1988	8th ICDA 1969-1978	7th ICD 1958-1968	6th ICD* 1950-1957
All Causes	1	001-999	000-999	001-999	
Infective and Parasitic Diseases	2	001-139	000-136	001-138,571, 696,697,764, 767,768, 785.6	
Neoplasms	3	140-239	140-239	140-219,222-239,292.3, 294	
Endocrine/Nutritional/Metabolic Diseases	4	240-279	240-279	250-289,772	
Blood Diseases	5	280-289	280-289	290-291, 292.0,.1,.2, .4,.5,.6,.7, 295-297, 298.1,299, 468.0,.1,.2	
Mental Disorders	6	290-319	290-315	300-324, 325.0,.1,.2, .3,.5,326, 688.1,781.5	
Nervous System/Sense Organ Disease	7	320-389	320-389	340-364,366-398,744.1, 765,780.3	
Circulatory Disease	8	390-459	390-458	330-334,400-467,468.3, 570.2	
Respiratory Disease	9	460-519	460-519	240,241,470-527,763	
Digestive Disease	10	520-579	520-577	530-561, 570.0,.1,.3, .4,.5,572-587,784.6	
Genito-urinary Disease	11	580-629	580-629	590-637, 789.1	
Complications of Pregnancy	12	630-676	630-678	640-687, 688.0,.2,.3, 689	

* ICD codes for the 6th ICD (1950-57) are the same as those for the 7th ICD(1958-68) unless otherwise indicated.
NE indicates the codes are not equivalent.

Conversion of ICD to LCDC Disease Codes.

Description	LCDC Code	9th ICD 1979-1988	8th ICDA 1969-1978	7th ICD 1958-1968	6th ICD* 1950-1957
Skin/Subcutaneous Tissue Disease	13	680-709	680-709	221,690-695, 698-709, 710.1,.3,.4, .5,711-716, 766	
Musculoskeletal Disease	14	710-739	710-738	710.0,720-743,744.0, .2,745-747, 749	
Congenital Anomalies	15	740-759	740-759	220,325.4, 710.2,748, 750-759	
Perinatal Mortality	16	760-779	760-779	760-762,769-771,773-776	
Symptoms/Ill-Defined	17	780-799	780-796	780.0,.1,.2, .4,.5,.6,.7, .8,.9,781.0, .1,.2,.3,.4, .6,.7,.8,.9, 782-783, 784.0,.1,.2, .3,.4,.5,.7, .8,785.0,.1, .2,.3,.4,.5, .7,.8,.9, 786-788, 789.0,.2,.3, .4,.5,.6,.7, .8,790-795	
Accidents/Poisonings/Violence	18	800-999	800-999	365,800-999	
Cancer	19	140-208	140-209	140-207, 292.3,294	
Lip	20	140	140	140	
Tongue	21	141	141	141	
Salivary Gland	22	142	142	142	
Mouth	23	143-145	143-145	143-144	
Oropharynx	24	146	146	145	

* ICD codes for the 6th ICD (1950-57) are the same as those for the 7th ICD (1958-68) unless otherwise indicated.
NE indicates the codes are not equivalent.

Conversion of ICD to LCDC Disease Codes.

Description	LCDC Code	9th ICD 1979-1988	8th ICDA 1969-1978	7th ICD 1958-1968	6th ICD* 1950-1957
Nasopharynx	25	147	147	146	
Hypopharynx	26	148	148	147	
Pharynx - Unspecified	27	149.0	149	148	
Pharynx *	28	146-148,149.0	146-149	145-148	
Buccal Cavity and Pharynx	29	140-149	140-149	140-148	
Tongue, Mouth and Pharynx	30	141,143-149	141,143-149	141,143-148	
Esophagus	31	150	150	150	
Stomach	32	151	151	151	
Small Intestine	33	152	152	152	
Colon	34	153,159.0	153	153	
Rectum and Rectosigmoid Junction	35	154	154	154	
Large Intestine and Rectum	36	153,154,159.0	153,154	153,154	
Liver	37	155.0,155.1	155	155.0	NE
Gallbladder	38	156	156	155.1	NE
Liver and Gallbladder	39	155.0,.1,156	155,156	155	
Pancreas	40	157	157	157	
Peritoneum	41	158	158	158	
Digestive - Unspecified	42	159.1,.8,.9	159	159	
Digestive	43	150-159	150-159	150-159	
Nose/Sinuses	44	160	160	160	
Larynx	45	161	161	161	
Bronchus and Lung	46	162.2,.3,.4, .5,.8,.9	162.1	162.1	NE
Pleura	47	163	163.0	162.2	NE

* ICD codes for the 6th ICD (1950-57) are the same as those for the 7th ICD (1958-68) unless otherwise indicated.
NE indicates the codes are not equivalent.

Conversion of ICD to LCDC Disease Codes.

Description	LCDC Code	9th ICD 1979-1988	8th ICDA 1969-1978	7th ICD 1958-1968	6th ICD* 1950-1957
Trachea/Bronchus/Lung and Other	48	162-163,164.2, .3,.8,.9,165	162,163	162-164	
Respiratory	49	160-163,164.2, .3,.8,.9,165	160-163	160-162,164	
Bone	50	170	170	196	
Connective Tissue	51	164.1,171	171	197	
Malignant Melanoma	52	172	172	190	
Melanoma Lip	53	172.0	172.0	190.0	NE
Melanoma Eyelid	54	172.1	172.1	190.1	NE
Melanoma Ear	55	172.2	172.2	190.2	NE
Melanoma Other Face	56	172.3	172.3	190.3	NE
Melanoma Scalp and Neck	57	172.4	172.4	190.4	NE
Melanoma Scrotum	58	187.7	172.5	179.1	NE
Melanoma Trunk	59	172.5	172.6	190.5	NE
Melanoma Upper Limb	60	172.6	172.7	190.6	NE
Melanoma Lower Limb	61	172.7	172.8	190.7	NE
Melanoma Site - Unspecified	62	172.8,.9	172.9	190.9	NE
Other Skin	63	173	173	191	
Breast	64	174,175	174	170	
Cervix Uteri	65	180	180	171	
Chorionepithelioma	66	181	181	NE	NE
Corpus Uteri	67	182	182.0	172	
Chorio. and Uterus - Unspecified	68	179,181	181,182.9	173,174	
Other Uterus	69	179,182	182	NE	NE
Chorio and Other Uterus	70	179,181,182	181,182	172-174	

* ICD codes for the 6th ICD (1950-57) are the same as those for the 7th ICD (1958-68) unless otherwise indicated.
NE indicates the codes are not equivalent.

Conversion of ICD to LCDC Disease Codes.

Description	LCDC Code	9th ICD 1979-1988	8th ICD 1969-1978	7th ICD 1958-1968	6th ICD* 1950-1957
Uterus Incl. Cervix	71	179-182	180-182	171-174	
Ovary	72	183.0	183.0	175.0	NE
Ovary/Fallopian Tube/Broad Ligament	73	183	183	175	
Female Genital	74	179-184	180-184	171-176	
Prostate	75	185	185	177	
Testis	76	186	186	178	
Male Genital	77	185,186,187.1, .2,.3,.4,.5, .6,.8,.9	185-187	177-179	
Kidney	78	189.0,.1,.2	189.0,189.1, 189.2	180	
Bladder	79	188	188	181.0	NE
Bladder and Other Urinary	80	188,189.3,.4, .8,.9	188,189.9	181	
Urinary	81	188,189	188,189	180,181	
Eye	82	190	190	192	
Brain	83	191	191	193.0	NE
Brain and Other Nervous System	84	191,192	191,192	193	
Nervous System Benign and Malignant	85	191,192,225, 237.5,237.6, 239.6	191,192,225, 238	193,223,237	
Thyroid	86	193	193	194	
Other Endocrine	87	164.0,194	194	195	
Ill-Defined Cancer	88	195-199	195-199	156,163,165, 198,199	
Lymphosarcoma and Retic.Cell Sarcoma	89	200.0,200.1	200	200.0,200.1	
Reticulum Cell Sarcoma	90	200.0	200.0	200.0	
Lymphosarcoma	91	200.1	200.1	200.1	

* ICD codes for the 6th ICD (1950-57) are the same as those for the 7th ICD(1958-68) unless otherwise indicated.
NE indicates the codes are not equivalent.

Conversion of ICD to LCDC Disease Codes.

Description	LCDC Code	9th ICD 1979-1988	8th ICDA 1969-1978	7th ICD 1958-1968	6th ICD* 1950-1957
Hodgkin's Disease	92	201	201	201	
Other Lymphoid	93	200.2,200.8, 202	202	200.2,202, 205	
Non-Hodgkin's Lymphomas	94	200,202	200,202	200,202,205	
Multiple Myeloma	95	203	203	203	
Lymphoid	96	200-203	200-203	200-203,205	
Lymphatic Leukemia	97	204	204	NE	NE
Myeloid Leukemia	98	205	205	NE	NE
Monocytic Leukemia	99	206	206	204.2	
Lymphatic and Myeloid Leukemia	100	204,205	204,205	204.0,204.1, 204.3	
Other and Unspecified Leukemia	101	207.0,.2,.8, 208	207	204.4	
Leukemia	102	204-206,207.0, .2,.8,208	204-207	204	
Polycythemia Vera	103	207.1,238.4	208	294	
Myelofibrosis	104	NE	209	292.3	
Neoplasms of Lymphatic and Hematopoietic Tissue	105	NE	200-209	200-205, 292.3,294	
Benign Neoplasms	106	210-229	210-228	210-219,222-229	
Neoplasm of Unspecified Nature	107	233.1,235-238	230-239	230-239	
Neoplasms, Secondary, Ill-Defined and Benign	108	195-199,210-229,233.1,235-238	195-199,210-239	156,163,165, 198,199,210-219,222-239	
Diabetes Mellitus	109	250	250	260	
Cystic Fibrosis	110	277.0	273.0	NE	NE
Aplastic Anemia	111	284	284	292.4	

* ICD codes for the 6th ICD (1950-57) are the same as those for the 7th ICD (1958-68) unless otherwise indicated.
NE indicated the codes are not equivalent.

Conversion of ICD to LCDC Disease Codes.

Description	LCDC Code	9th ICD 1979-1988	8th ICDA 1969-1978	7th ICD 1958-1968	6th ICD* 1950-1957
Alcoholic Psychosis, Alcoholism and Alcohol Poisoning	112	291,303,305.0, 860	291,303,860	307,322,880	
Multiple Sclerosis	113	340	340	345	
Paralysis Agitans	114	332.0	342	350	
Motor Neurone Disease	115	335.2	348	356.0,.1,.3	
Major Cardio-Vascular Diseases	116	390-448	390-448	330-334,400-456,467.1, 570.2	
Ischemic Heart Disease	117	410-414	410-413	420	
Cerebrovascular Disease	118	430-438	430-438	330-334	
Heart Disease	119	NE	391,392.0, 393-398,402, 404,410-429	401,402.1, 410-443	NE
Rheumatic Fever and Chronic Rheumatic Heart Disease	120	390-398,424.0, .1,.3	390-398	400-402,410-416	
Hypertensive Disease	121	401-405	400-404	NE	NE
Active Rheumatic Fever	122	390-392	390-392	400-402	
Chronic Rheumatic Heart Disease	123	393-398,424.0, .1,.3	393-398	410-416	
Acute Myocardial Infarction	124	410	410	NE	NE
Other Forms of Heart Disease	125	NE	420-429	NE	NE
Diseases of Arteries, Arterioles and Capillaries	126	440-448	440-448	450-456, 467.1,570.2	
Aortic Aneurysm	127	441.0,.1,.2, .3,.4,.5.,.6	441	451	
Diseases of Veins, Lymphatics and Other Disease of Circulatory System	128	415.1,451-459	450-458	460-467, 468.3	
Acute Respiratory Infections, Except Influenza	129	460-466	460-466	470-475,500	
Influenza	130	487	470-474	480-483	

* ICD codes for the 6th ICD (1950-57) are the same as those for the 7th ICD (1958-68) unless otherwise indicated.
NE indicates the codes are not equivalent.

Conversion of ICD to LCDC Disease Codes.

Description	LCDC Code	9th ICD 1979-1988	8th ICDA 1969-1978	7th ICD 1958-1968	6th ICD* 1950-1957
Pneumonia	131	480-486	480-486	490-493, 763	
Chronic Bronchitis, Emphysema and Asthma	132	490-493 496	490-493, 519.3	241,501, 502,527.1	
Other Diseases of Upper Respiratory Tract	133	470-478	500-508	240, 510-517	
Other Diseases of Respiratory System	134	494,495 497-519	510-518, 519.0,.1,.2, .9	518-526, 527.0,527.2	
Viral Pneumonia	135	480	480	NE	NE
Pneumococcal Pneumonia	136	481	481	NE	NE
Other Bacterial Pneumonia	137	482	482	NE	NE
Pneumonia due to Other Specified Organism	138	483	483	NE	NE
Other Pneumonia	139	484-486	484-486	NE	NE
Pneumoconiosis	140	011.4,495, 500-505	010,515,516	001,523,524	
Silicosis and Anthracosilicosis	141	500,502	515.0,515.1	523.0,523.1	
Asbestosis	142	501	515.2	523.2	
Other Pneumoconiosis	143	495,503-505	515.9,516	523.3,524	
Silicotuberculosis	144	011.4	010	001	
Ulcer of Stomach	145	531	531	540	
Ulcer of duodenum	146	532	532	541	
Ulcers	147	531-534	531-534	540-542	
Cirrhosis of Liver	148	571	571	298.0,581	
Gallbladder Diseases	149	574-576	574-576	584-586	
Diseases of Pancreas	150	577	577	587	

* ICD codes for the 6th ICD (1950-57) are the same as those for the 7th ICD(1958-68) unless otherwise indicated.
NE indicates the codes are not eqivalent.

Conversation of ICD to LCDC Disease Codes.

Description	LCDC Code	9th ICD 1979-1988	8th ICDA 1969-1978	7th ICD 1958-1968	6th ICD* 1950-1957
Nephritis and Nephrosis	151	580-585,587	580-584	590-594	
Kidney Infections	152	590	590	600	
Kidney Diseases	153	580-593	580-593	590-603, 789.1	
Prostate Disease	154	222.2,600-602	600-602	610-612	
Congenital Anomalies of Nervous System	155	740-742	740-743	750-753	
Congenital Anomalies of Circulatory System	156	745-747	746-747	754	
Motor Vehicle Traffic Accidents	157	810-819	810-819	810-825	
MVTA Involving Pedestrian	158	814	814	812	
Accidental Falls	159	880-888	880-887	800-904	
Accidents Caused by Fires and Flames	160	890-899	890-899	916	
Accidents Caused by Fire in Private Dwelling	161	890	890	NE	NE
Accidental Drownings	162	910	910	929	
Suicide	163	950-959	950-959	970-979	
Homicide	164	960-969	960-969	980-983	
Chronic Respiratory Disease	165	490-508, 515-517	490-493,515-518,519.2, 519.3	241,501,502, 523-526, 527.1	
Cirrhosis and Other Liver Disease	166	570-573	570-573	298.0,580-583	
Prostatitis Neoplasia and Hyperplasia	167	185,222.2, 233.4,236.5, 600	185,600	177,610	
Prostatitis and Other Diseases of Prostate	168	601,602	601,602	611,612	
All Natural Causes of Death	169	001-799	000-796	001-795	

* ICD codes for the 6th ICD (1950-57) are the same as those for the 7th ICD (1958-68) unless otherwise indicated.
NE indicates the codes are not equivalent.

Conversion of ICD to LCDC Disease Codes.

Description	LCDC Code	9th ICD 1979-1988	8th ICDA 1969-1978	7th ICD 1958-1968	6th ICD* 1950-1957
Appendicitis	170	540-543	540-543	550-553	
Renal Calculi	171	592,594	592,594	602-604	
Accidental Poisoning	172	E850-869	E850-877	E870-895	
Asthma	173	493	493	241	
Chronic Obstructive Pulmonary Disease	174	490-496	NE	NE	NE

* ICD codes for the 6th ICD (1950-57) are the same as those for the 7th ICD(1958-68) unless otherwise indicated.
NE indicates the codes are not equivalent.

APPENDIX C

DESCRIPTIVE TABLES

Years Since First Exposure vs. Vital Status

YSFE VITAL STATUS

Frequency / Row Pct	ALIVE	DEAD	Total
0- 0.9	139 98.58	2 1.42	141
1- 9.9	954 96.07	39 3.93	993
10-19.9	879 91.75	79 8.25	958
20-29.9	1223 89.47	144 10.53	1367
30-39.9	970 84.64	176 15.36	1146
40.0+	412 50.93	397 49.07	809
Total	4577	837	5414

Distribution of Calendar Period of Death

DEATHY	Frequency	Percent
1950-54	31	3.7
1955-59	67	8.0
1960-64	90	10.8
1965-69	90	10.8
1970-74	121	14.5
1975-79	126	15.1
1980-84	164	19.6
1985-89	148	17.7

Distribution of Age at Death

AGEDEATH	Frequency	Percent
20-24.9	8	1.0
25-29.9	8	1.0
30-34.9	21	2.5
35-39.9	19	2.3
40-44.9	49	5.9
45-49.9	54	6.5
50-54.9	80	9.6
55-59.9	98	11.7
60-64.9	97	11.6
65-69.9	105	12.5
70-74.9	94	11.2
75-79.9	86	10.3
80-84.9	58	6.9
85.0+	60	7.2

Distribution of Length of Follow-up

FOLLOWUP	Frequency
0- 0.9	144
1- 9.9	1079
10-19.9	1090
20-29.9	1523
30-39.9	1182
40.0	396

Distribution of Birth Year

BIRTH YEAR	Frequency	Percent
pre 1900	320	5.9
1900-09	152	2.8
1910-19	188	3.5
1920-29	742	13.7
1930-39	1355	25.0
1940-49	1141	21.1
1950-59	1067	19.7
1960+	449	8.3

Distribution of Hire Year

HIRE YEAR	Frequency	Percent
pre 1900	3	0.1
1900-09	21	0.4
1910-19	179	3.3
1920-29	184	3.4
1930-39	120	2.2
1940-49	522	9.6
1950-59	1112	20.5
1960-69	1284	23.7
1970-79	893	16.5
1980-89	1096	20.2

Distribution of Death Year

DEATH YR	Frequency
1950-54	31
1955-59	67
1960-64	90
1965-69	90
1970-74	121
1975-79	126
1980-84	164
1985-89	148
TOTAL	837

Distribution of Age at Hire

AGE AT HIRE	Frequency	Percent
15-19.9	62	1.1
20-24.9	2900	53.6
25-29.9	2072	38.3
30-34.9	268	5.0
35-39.9	51	0.9
40-44.9	29	0.5
45-49.9	15	0.3
50-54.9	11	0.2
55-59.9	5	0.1
60.0+	1	0.0

Distribution of Years Worked

Years Worked	Frequency	Percent
MISSING	41	0.8
0- 0.9	181	3.3
1- 4.9	606	11.2
5- 9.9	677	12.5
10-14.9	608	11.2
15-19.9	510	9.4
20-24.9	697	12.9
25-29.9	737	13.6
30-34.9	842	15.6
35.0+	515	9.5

Distribution of Time Between Termination and Death

TIME FROM TERMINATION UNTIL DEATH	Frequency	Percent
0-7 days	200	23.9
8-14 days	2	0.2
15 days-1 month	3	0.4
1-6 months	30	3.6
6 months- 1 Year	23	2.7
1-2 Years	22	2.6
more than 2 Years	533	63.7

APPENDIX D
MEMO ON INDEX OF EXPOSURE FOR FIRE FIGHTERS

Date: May 15, 1991

Memo to file: From K.A. L'Abbé G.A. Tomlinson

Re: Exposure Index in Metropolitan Toronto fire fighter's Study

---

For the IDSP's cohort study of fire fighters, it would be useful to have an index of exposure which could be related to the mortality of the fire fighters, and in this way, an exposure-response relationship could be investigated. Since the design of this study, much thought has been put into an appropriate exposure index. The extent to which the occupational environment of the fire fighter can be characterized is limited by the availability of data. In the best of all possible worlds, fire fighters would wear personal monitoring devices which would measure several chemicals and combustion products each time they responded to an alarm. Recently, this type of monitoring has only taken place when specific (and small) studies have been undertaken (Brandt-Rauf et al., 1988). Short of this, there are several possibilities for exposure data.

In this memo, the first section will review the exposure indices used in cohort studies of fire fighters, concluding with possibilities for our study of fire fighters. In the second section, I will discuss the possible exposure and indices which could be used if a nested case-control study is undertaken in the future within this cohort of fire fighters.

Section 1:

The following studies report the mortality or cancer incidence of fire fighters with no exposure index:

Mastromatteo, 1959 (Toronto)
Musk et al., 1978 (Boston)
Sardinas et al., 1986 (Connecticut)
Rosenstock et al., 1990 (Washington State)
Sama et al., 1990 (Massachusetts)
Hanson, 1990 (Denmark)

Several studies simply use number of years (duration) of employment as a fire fighter as the surrogate for exposure. Use of duration as the exposure index is most valid under circumstances in which the dose rate has remained constant over the entire employment period for each fire fighter. The following studies of fire fighters used duration (years) employed as a fire fighter as the only exposure index:

Eliopulos et al,. 1984 (Australia)

Vena and Fiedler, 1987 (Buffalo)
Heyer et al., 1990 (Seattle)
Beaumont et al., 1991 (San Francisco)

In addition to duration of employment, three studies of fire fighters have attempted to characterize "exposure opportunity". In Guidotti (Edmonton and Calgary, 1991) (not yet published), duration of employment is multiplied by a weight and the result is the "exposure opportunity index". The weights are 1 = fire fighter, 0.2 = safety or training officer, 0.1 = captain, 0 = desk job. [Heyer et al., 1990 also do this implicitly by assigning zero duration of employment to employees of fire departments who are not assigned to active fire combat duty.] Guidotti also attempted to develop an index based on fire hall activity, representing the number of real alarms (as opposed to false alarms) and the proportion of those representing fires. However, there were (was?) "insufficient data and job assignment by hall... to construct an meaningful profile based on activity levels. Fire halls vary in their level of activity in each city, but rotation among halls is common in modern, urban, professional fire services" (pp 6-7). Therefore this index of fire hall activity was not used.

Bates (1987) Toronto, also attempted to use a "relative index of fire-fighting activity". For each of the approximately 30 fire halls within the Toronto Fire Department, the number of calls per fire hall was available in the period 1970 to 1984, and the assumption was made that the relative activity would apply to the fire halls before 1970. Bates thought that records were available on the assignment of each fire fighter to fire halls along with duration, and a scale was developed to indicate fire-fighting activity with 1 = high, 2 = medium and 3 = low activity. However, no results are reported by this index, and Bates says, "the creation of a relative scale of fire-fighting activity was successful, but the subsequent utilization was problematic; it was impossible to determine from the record the activity level for nearly one-third of the fire fighters who died." Further, "... posting to an active hall doesn't necessarily lead to an active individual effort, because missing data caused a lack of power or because no relationship [between fire-fighting activity and coronary artery disease death] exists."

Finally, in addition to duration of employment, investigators in one other study attempted an index of "presumed chemical exposure" (Feuer and Rosenman, New Jersey, 1986). "Because individual measures of exposure to chemicals did not exist, a crude surrogate measure was developed. The chemical index was derived by calculating the percentage of the work force employed in the chemical industry in 1958, by municipality, using data supplied by the New Jersey Department of Labor and Industry... either light (2% or less), moderate (2-5%) or heavy (greater than 5%) concentration of chemical industry employment."

In this proportional mortality study, a relationship was seen between this index and cirrhosis of the liver only.

In terms of the IDSP's study of fire fighters, records were not consistently available which contained information of fire hall posting for individual fire fighters. Although fire hall activity is available for almost two decades in most departments, individual assignments to fire halls, along with lack of data on the knowledge that considerable rotation occurs among fire halls by fire fighters, an index to fire hall activity (and therefore "exposure opportunity") is not possible for this study. At the level of the fire department (there are six departments within this study of Metropolitan Toronto), it is certainly possible to develop an index of fire department activity, however, two major problems arise. First, within a department each fire fighter would be assigned the same level of activity and lack of exposure variability would result in not being able to see an exposure-response relationship if one exists. The index would virtually vary only by duration of employment. Second, an individual's exposure opportunity may or may not be reflected through the department's level of activity: the exposure misclassification which would be expected using this method would severely dilute the chances of discerning an exposure-response relationship if one exists. Further, the type of fire and the dollar damage, etc. is available at the fire department level, but the use of this information suffers from the same exposure misclassification problems.

As in several recent studies of fire fighters, number of years (duration) of employment will be used in the IDSP's study. It is very difficult to know how well duration measures actual exposure. For example, with the advent of plastics in the 1950's and their increasing use since then, toxic exposure of this nature would have likely increased over the last three decades. However, protective equipment has improved over this time, and it is impossible to know how all the various factors interact and to what extent an assumption of constant exposure over time could be valid.

In terms of weighing duration of employment by fire fighter status, according to job at hire in our cohort, about 75% of the fire department employees are fire fighters. However, of the remaining 25%, only 5% are known to have other duties, such as training officer. For the remaining 20%, the records did not specify the job at hire, and from discussions with fire department officials, most of those with missing information are fire fighters. Given that the majority of our cohort are fire fighters, weighting of duration will yield results very similar to those from the use of duration itself.

Section 2:

As planned in the protocol, if an excess of a specific cause of death is apparent from the cohort study, a case-control study of this cause may be undertaken. In addition to information from department records, information from personal interviews can lead to the collection of very detailed exposure information, such as:

- circumstances and activities at fires
- use of protective equipment, specifically self-contained breathing equipment
- training experience
- demographic and health-related information
- perception of smoke intensity
- structural materials and burning materials of major incidence
- occurrence of death of colleagues or others at fires (critical incident stress)

However, by definition (since mortality was the outcome investigated in the cohort study) cases are deceased; therefore, proxies must be interviewed. For cases, demographic and health-oriented information (smoking, etc.) can be gathered from next-of-kin. For work-related factors, appropriate proxies are probably co-workers, thus raising the problem of two sources of information for each case. The same method of data collection could be followed for controls, with an additional methodological element of interviewing controls directly for all variables to check concordance with the proxies.

Conclusion:

In the absence of information on individual fire fighter activity and fire hall assignment and in light of the small variation in fire fighter job assignment, the only feasible index of exposure for the cohort study is number of years of employment. In this respect, the results will be directly comparable to those from other studies. For this to be a valid approximation to the exposure to fires experienced by fire fighters, actual exposure should be close to uniform over a fire fighter's career, over calendar time and across fire fighters. Any departure from this uniformity will be masked in the analysis by the use of years of employment as the sole index of exposure. As a result, variability in risk associated with variability in exposure will go undetected and risks associated with fire fighting may be underestimated.

If a case-control study is undertaken, the smaller study population involved will make it practicable to generate an index of exposure based on personal interviews. This will provide much stronger power to detect increased risk of disease associated with fire fighting.

APPENDIX E
OCCUPATIONAL EXPOSURES OF FIRE FIGHTERS IN METROPOLITAN MONTREAL

OCCUPATIONAL EXPOSURES OF FIRE FIGHTERS IN THE METROPOLITAN MONTREAL

by Louise Nadon, Ramzan Lakhani, and Jack Siemiatycki

The following report was prepared for the Ontario Industry Disease Standards Panel to be used in the exposure assessment of a cohort study of fire fighters in Metropolitan Toronto. We were specifically requested to provide detailed information on fire fighters (guesstimates of exposure and relative magnitude of these exposures) extracted from our large population-based case-control study of cancer and to discuss the relevance of this Montreal data for Toronto fire fighters.

The cohort study under way in Toronto spans the last 30 years and the relevance of our data for this time period can be judged by consulting Table 1 which lists all the jobs included in this report sorted by the date on job entry. One can also see from the Standard Industrial Classification code assigned to each of these jobs, that 40 out of 47 were urban fire-fighters. The relevance of data collected in Metropolitan Montreal to a study carried out among Toronto fire fighters depends on the nature or origin of fires in both these cities. In effect, fires in private residences, offices and commercial centers should be similar in both cities and lead to similar occupational exposures. Differences could occur if one or either city had a high proportion of industrial fires leading to very specific occupational exposures (e.g. a fire in a chemical plant). The 1990 annual report of the City of Montreal's Fire Department provided us with data on to the various causes and origins of fires in Montreal for that year (and limited information for 1988 and 1989). In 1990, most fires in the city of Montreal occurred in private residences or in commercial establishments; in fact none of the major fires occurring during that year were of an industrial nature.

Results of the assessment of occupational exposures of the fire fighters in our dataset are presented in table 2. The data is sorted in decreasing order of the prevalence of the exposure among all fire fighters in our study. Our evaluation of an exposure is always assessed according to the four following dimensions: contact, concentration, frequency and reliability. Each can take on a value of 1, 2 or 3. Contact 1 is coded when the subject is exposed to the pollutant by inhalation only, contact 2 refers exposures which occur only through contact with the skin and contact 3 is coded when the exposure is both inhaled and cutaneous. Concentration refers to the intensity of the exposure and is also coded as 1, 2 and 3 for low, medium and high concentrations respectively. The frequency of the exposure refers to the proportion of work time during which the subject is exposed to the pollutant, frequency 1 is coded when the exposure occurs less than 5% of the time, frequency 2 corresponds to

between 5 and 30% and frequency 3 is coded when the exposure occurs more than 30% of the work time. The reliability index gives an indication of the chemist's/hygienist's confidence that such an exposure actually occurred, reliability 1 indicate

s that the exposure is possible but not probable, reliability 2, that the exposure is probable and reliability 3 that the exposure was definite. In table 2, the results are broken down according to these three dimensions of exposure. Most urban firemen would most likely be exposed to the most prevalent exposures in this table, i.e. those at the beginning of the table and these are the exposures which could be included in a job exposure matrix for this occupation. We must caution the reader against using the less prevalent exposures in such a matrix as these probably are due to some idiosyncrasy in the individual's work history and most fire fighters are probably not exposed to these pollutants.

Several exposures mentioned in the literature, for example, trichloroethylene, perchloroethylene, chloroform, methylene chloride, toluene and trichlorophenol appearing in a publication by Brandt-Rauf (5) are not included in our exposure assessment since it was felt that the intensity and/or frequency of these exposure were to low to warrant inclusion. Some exposures may have been overlooked, either because they are not part of the list of some 300 chemicals which we evaluate routinely or because we have seen no indication in the literature to support their inclusion.

Table 1

Jobs classified as firefighters (CCDO occupational code 6111) in our dataset sorted by date on job entry

Matjob	Dates		SIC Industry code*	CCDO Occupational code**	
88701	28	74	503	6111	126
50102	38	73	951	6111	126
54502	38	70	951	6111	126
223702	38	74	951	6111	126
32101	41	71	951	6111	126
216304	41	55	951	6111	126
248502	43	73	951	6111	126
462303	43	47	951	6111	126
702301	43	46	951	6111	126
70401	44	74	951	6111	126
309003	45	76	951	6111	126
596402	45	74	951	6111	126
81601	46	76	951	6111	126
402904	46	59	951	6111	126
459902	46	72	951	6111	126
512702	46	77	951	6111	126
41503	48	58	503	6111	114
418606	48	53	951	6111	126
530102	49	78	951	6111	126
545001	50	81	951	6111	126
79405	51	79	951	6111	126
105204	51	76	951	6111	126
848903	51	80	951	6111	126
414602	52	54	951	6111	126
429403	52	83	951	6111	126

Matjob	Dates		SIC Industry code*	CCDO Occupational code**	
83602	53	71	902	6111	118
412502	53	70	951	6111	126
505702	53	83	951	6111	126
823705	54	85	951	6111	126
12102	56	79	951	6111	126
231702	56	81	951	6111	126
411505	56	82	951	6111	126
597802	56	60	902	6111	122
39802	57	73	951	6111	126
41504	58	79	951	6111	118
247605	60	70	951	6111	126
436502	60	83	951	6111	126
248003	61	81	951	6111	126
456703	61	83	951	6111	126
579902	63	84	951	6111	126
517003	66	84	951	6111	126
871803	67	85	502	6111	126
309203	68	81	951	6111	126
582406	68	77	327	6111	118
83603	71	80	909	6111	114
39803	73	79	951	6111	114
105205	76	80	951	6111	126

* SIC 327 = Shipbuiding and repair
503 = Railway transport
909 = Other federal administration
951 = Local administration

** CCDO 6111-114= Fire captain
6111-118 = Fire chief
6111-122 = Fire fighter, crash
6111-126 = Fire fighter

Table 2

EXPOSURES OF FIREFIGHTERS IN THE MONTREAL AREA

Chemical code	Exposure	Prevalence	# of jobs	# jobs with contact 1	2	3	# jobs with conc. 1	2	3	# jobs with freq. 1	2	3	# jobs with reliab. 1	2	3	Remarks	References
530194	PAH's from other sources	97.9	46	46	0	0	2	8	36	17	29	0	0	2	44		
370001	Other pyrolysis fumes	95.7	45	45	0	0	1	9	35	16	29	0	0	1	44		
520599	Aliphatic aldehydes	95.7	45	45	0	0	7	38	0	15	30	0	0	2	43	Formaldehyde, acrolein, etc...	2, 5, 6, 13, 14, 17, 19, 22
210601	Carbon monoxide	93.6	44	44	0	0	2	6	36	14	29	1	0	1	43	Formed during incomplete combustion processes	5, 6, 8, 14, 21, 22
220501	Formaldehyde	93.6	44	44	0	0	7	37	0	15	29	0	0	2	42	Formed during combustion of most organic compounds	2, 5, 14, 19
110029	Ashes	91.5	43	43	0	0	6	37	0	15	28	0	0	3	40		
170004	Soot	91.5	43	1	0	42	40	3	0	39	4	0	0	3	40		
210703	Nitrogen oxides	91.5	43	43	0	0	3	35	5	14	28	1	0	0	43	Combustion of nitrogen containing compounds	6, 8, 13, 21, 22
370009	Wood combustion	91.5	43	43	0	0	2	5	36	14	29	0	0	2	41		
411603	Sulphuric acid	91.5	43	43	0	0	43	0	0	40	3	0	2	39	2	Formed during combustion of sulfur containing compounds	8
520899	Chlorinated alkanes	91.5	43	4	0	39	2	41	0	40	3	0	1	34	8	Fire extinguishers and dry-cleaning shops	3, 16
530195	PAH's from wood	91.5	43	43	0	0	2	5	36	14	29	0	0	2	41		
530196	PAH's from petroleum	91.5	43	29	0	14	1	42	0	9	34	0	0	0	43	Gasoline, solvents	
530198	Benzo(a)pyrene	91.5	43	43	0	0	6	37	0	14	29	0	0	2	41		
110007	Chrysotile asbestos	89.4	42	42	0	0	42	0	0	13	29	0	0	3	39	Heat insulation, fireproof clothing	2, 3
211601	Sulphur dioxide	89.4	42	42	0	0	36	6	0	12	30	0	0	8	34		5, 13
530199	MAH's	89.4	42	34	0	8	8	33	1	11	31	0	0	5	37		2, 3, 5, 6, 9, 22
211700	Chlorine	87.2	41	41	0	0	8	32	1	13	28	0	1	6	34	Pyrolysis of Polyvinyl chloride products	8, 21

Chemical code	Exposure	Prevalence	# of jobs	# jobs with contact 1	2	3	# jobs with conc. 1	2	3	# jobs with freq. 1	2	3	# jobs with reliab. 1	2	3	Remarks	References
225001	Phosgene	87.2	41	41	0	0	5	36	0	40	1	0	0	31	10	Heating of chlorinated hydrocarbons	4, 8, 21
370004	Engine emissions	87.2	41	41	0	0	3	38	0	9	32	0	0	0	41		
370006	Diesel engine emissions	87.2	41	41	0	0	4	37	0	11	30	0	0	6	35		
370014	Rubber pyrolysis products	87.2	41	41	0	0	4	36	1	12	29	0	0	34	7	Carpet backing, tires	
420801	Carbon tetrachloride	87.2	41	0	0	41	4	37	0	40	1	0	2	34	5	Pyrene fire extinguishers	4
460003	Solvents	87.2	41	0	0	41	26	15	0	35	6	0	0	9	32		11
460004	Waxes, polishes	87.2	41	0	0	41	39	2	0	39	2	0	1	9	31	Polishing fire truck	
520199	Alkanes (C5-C17)	87.2	41	0	0	41	26	15	0	36	5	0	0	9	32		9, 11, 21
990005	Cleaning agents	87.2	41	0	0	41	0	40	1	4	37	0	0	3	38	Cleaning fire truck	
210602	Hydrogen cyanide	85.1	40	40	0	0	4	35	1	12	28	0	1	2	37	Mostly formed during combustion of synthetics	5, 6, 8, 9, 13, 14, 17, 19, 21,
210701	Ammonia	85.1	40	40	0	0	5	35	0	12	28	0	1	32	7	Mostly formed during combustion of synthetics	8, 9, 19, 22
211602	Hydrogen sulphide	85.1	40	40	0	0	4	36	0	12	28	0	0	6	34		8, 19
211701	Hydrogen chloride	85.1	40	40	0	0	4	35	1	12	28	0	1	2	37		5, 6, 7, 13, 14, 17, 19, 21, 22
370013	Plastics pyrolysis products	85.1	40	40	0	0	0	7	33	12	28	0	1	3	36		9, 17, 18
430101	Benzene	85.1	40	40	0	0	5	34	1	11	29	0	1	5	34		2, 3, 5, 6, 9, 22
460012	Lubricating oils and	85.1	40	0	40	0	37	3	0	36	4	0	1	27	12		
510001	Cyanides	85.1	40	40	0	0	4	33	3	12	28	0	1	2	37	Mostly formed during combustion of synthetics	3, 6, 8, 9, 13, 14, 19, 21
520197	Alkanes (C18+)	85.1	40	0	7	33	35	5	0	36	4	0	0	26	14	Lubricating oils and greases, polishes	4
521699	Fluorocarbons	85.1	40	37	0	3	3	37	0	40	0	0	0	38	2	Halon fire extinguishers	4, 11
110014	Brick dust	83.0	39	39	0	0	39	0	0	39	0	0	0	39	0	Demolition, very low exposure	
110016	Concrete dust	83.0	39	39	0	0	39	0	0	39	0	0	0	39	0		

Chemical code	Exposure	Prevalence	# of jobs	# jobs with contact 1	2	3	# jobs with conc. 1	2	3	# jobs with freq. 1	2	3	# jobs with reliab. 1	2	3	Remarks	References
112002	Calcium sulphate	83.0	39	39	0	0	8	31	0	37	2	0	1	3	35	Gypsum board	
460027	Mineral spirits pre 1970	83.0	39	0	0	39	30	9	0	37	2	0	1	11	27		
518299	Lead compounds	83.0	39	39	0	0	38	1	0	8	31	0	0	1	38	Paints, engine emissions	
110002	Inorganic insulation dust	80.9	38	38	0	0	38	0	0	10	28	0	0	2	36	Demolition, very low exposure	
512699	Iron compounds	80.9	38	38	0	0	37	1	0	36	1	1	0	35	3	Fumes from metal structures	
430104	Styrene	78.7	37	37	0	0	6	30	1	9	28	0	0	34	3		9, 21
460009	Mineral spirits post 1970	78.7	37	0	0	37	29	8	0	36	1	0	2	7	28		
110008	Amphibole asbestos	76.6	36	36	0	0	36	0	0	34	2	0	1	35	0	Impurety in chrysotile asbestos	2, 3
513399	Arsenic compounds	76.6	36	36	0	0	32	4	0	36	0	0	1	35	0	Treated wood	2
310004	Metal oxide fumes	74.5	35	35	0	0	34	1	0	34	0	1	0	31	4		
531899	Isocyanates	74.5	35	35	0	0	32	3	0	32	3	0	0	32	3		9, 10, 19
990021	Biocides	63.8	30	30	0	0	28	2	0	30	0	0	2	28	0		
530193	PAH's from any source	47.0	33	0	14	14	32	1	11	36	0	2	1	44	0		
370008	Liquid fuel combustion	21.3	10	10	0	0	0	2	8	2	8	0	0	4	6	Combustion of heating fuel	
410003	Javel water	6.4	3	0	0	3	0	3	0	0	3	0	0	0	3	Used for cleaning duties around fire house	
460006	Kerosene	6.4	3	0	0	3	0	3	0	2	1	0	0	0	3		
470005	Metal coatings	6.4	3	2	0	1	0	3	0	2	1	0	0	0	3		
510004	Hypochlorites	6.4	3	0	0	3	0	3	0	0	3	0	0	0	3		
110001	Abrasives dust	4.3	2	2	0	0	1	1	0	1	1	0	0	1	1		
110005	Metallic dust	4.3	2	2	0	0	1	1	0	0	1	1	0	1	1		
110021	Mild steel dust	4.3	2	2	0	0	1	1	0	0	1	1	0	1	1		

Chemical code	Exposure	Prevalence	# of jobs	# jobs with contact 1	2	3	# jobs with conc. 1	2	3	# jobs with freq. 1	2	3	# jobs with reliab. 1	2	3	Remarks	References
460008	Heating oil	4.3	2	0	0	2	0	2	0	1	1	0	0	0	2		
460019	Hydraulic fluid	4.3	2	0	0	2	0	2	0	2	0	0	0	0	2		
530399	Aromatic amines	4.3	2	2	0	0	0	2	0	2	0	0	0	0	2		
110024	Inorganic pigments	2.1	1	0	0	1	1	0	0	0	1	0	0	0	1		
110026	Extenders	2.1	1	0	0	1	1	0	0	0	1	0	0	0	1		
112601	Iron oxides	2.1	1	0	0	1	1	0	0	0	1	0	0	1	0		
118[illegible]	Lead chromate	2.1	1	0	0	1	1	0	0	0	1	0	0	1	0		
150022	Alkyds	2.1	1	0	0	1	0	1	0	0	1	0	0	1	0		
210801	Ozone	2.1	1	1	0	0	1	0	0	0	0	1	0	1	0		
310001	Gas welding fumes	2.1	1	1	0	0	0	1	0	0	1	0	0	0	1		
310002	Arc welding fumes	2.1	1	1	0	0	0	1	0	0	0	1	0	0	1		
440002	Turpentine	2.1	1	0	0	1	0	1	0	0	1	0	0	0	1		
460005	Leaded gasoline	2.1	1	1	0	0	1	0	0	1	0	0	0	1	0		
460007	Diesel oil	2.1	1	0	0	1	0	1	0	1	0	0	0	0	1		
510003	Chromium (VI)	2.1	1	0	0	1	1	0	0	0	1	0	0	1	0		
512499	Chromium compounds	2.1	1	0	0	1	1	0	0	0	1	0	0	1	0		

BIBLIOGRAPHY

1. Bates JT. Coronary artery disease deaths in the Toronto Fire Department. J Occup Med 1987;29(2):132-135.

2. Beaumont JJ, Chu GST, Jones JR, et al. An epidemiologic study of cancer and other causes of mortality in San Francisco firefighters. Am J Ind Med 1991;19:357-372.

3. Bendix S. Firefighter exposure to environmental carcinogens. J Combustion Toxicology 1979;6:127-135.

4. Brady GS, Clauser HR. Materials Handbook (11th ed.). New York: McGraw-Hill, 1977.

5. Brandt-Rauf PW, Fallon LF Jr., Tarantini T, Idema C, Andrews L. Health hazards of fire fighters: exposure assessment. Br J Ind Med 1988;45:606-612.

6. Burgess W, Treitman RD, Gold A. Air Contaminants in Structural Firefighting. Final Report. Prepared for: National Fire Prevention and Control Administration. The Society of the Plastics Industry, Inc., 1979.

7. Chemical and Engineering News. Researchers providing some details about hazards of fire. Chemical and Engineering News May 20, 1985:35-38.

8. Dornette WHL. Synthetics and Fire . unpublished.

9. Farhi R, Morel C, Chéron J. Matières Plastiques et Adjuvants. Hygiène et Sécurité. Paris: Institut National de Recherche et de Sécurité, 1982.

10. Giguere MT, Hughes LD, Curran JP, Griggs T, Simpson J Ross J., Lohr CB. Positive pressure ventilation with fire suppression: Effective reduction of toxic gas exposures. Appl Occup Environ Hyg 1990;5(5):293-297.

11. Gosselin RE, Hodge HC, Smith RP, Gleason MN. Clinical Toxicology of Commercial Products: Acute Poisoning (4th ed.). Baltimore: Williams and Wilkins, 1976.

12. Hansen ES. A Cohort Study on the Mortality of Firefighters. British Journal of Industrial Medicine 1990;47(12):805-809.

13. Hartzell GE, Packham SC, Switzer WG. Toxic products from fires. Am Ind Hyg Assoc J 1983;44:248-255.

14. Lowry WT, Juarez L, Petty CS, Roberts B. Studies of toxic gas production during actual structural fires in the Dallas area. J Forensic Sci 1985;30(1):59-72.

15. Markowitz JS, Gutterman EM, Schwartz S, Link B, Gorman SM. Acute health effects among firefighters exposed to a polyvinyl chloride (PVC) fire. Am J Epidemiol 1989;129(5):1023-1031.

16. Patty FA. Industrial Hygiene and Toxicology. Vol.II. Toxicology. (2nd (Revised) ed.). New York: John Wiley and Sons, 1963.

17. Rawls R. Fire toxicity film angers plastics industry. C&EN September 9, 1985:25.

18. Rawls RL. Fire hazards of plastics spark heated debate. C&EN; January 3, 1983:9-16.

19. Rosenstock L, Demers P, Heyer NJ, Barnhart S. Respiratory mortality among firefighters. Br J Ind Med 1990;47:462-465.

20. Service de prévention des incendies de la ville de Montréal. Bilan des activites 1990. Rapport annuel 1990.

21. Sumi K, Tsuchiya Y. Toxic gases and vapours produced at fires. Canadian Building Digest 1971;144:1-4.

22. Treitman RD, Burgess WA, Gold A. Air contaminants encountered by firefighters. Am Ind Hyg Assoc J 1980;41:796-802.